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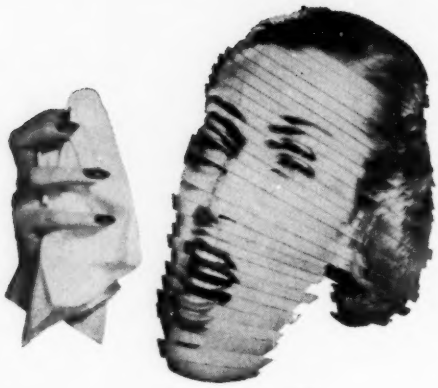

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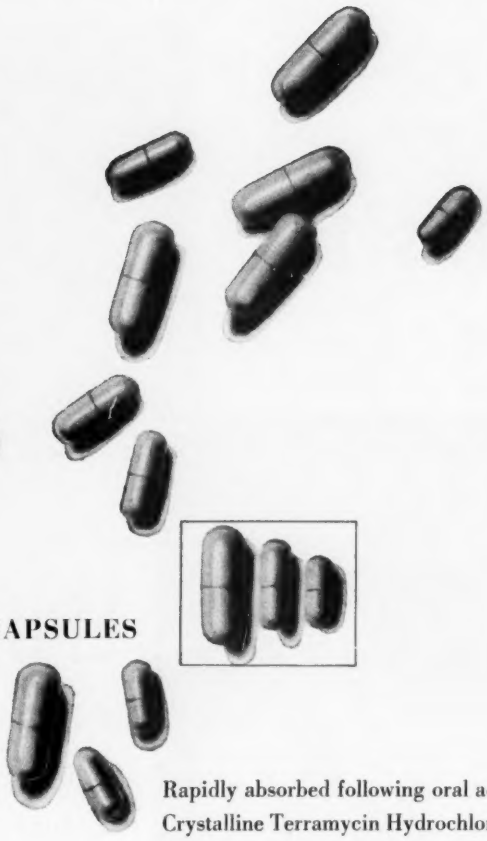

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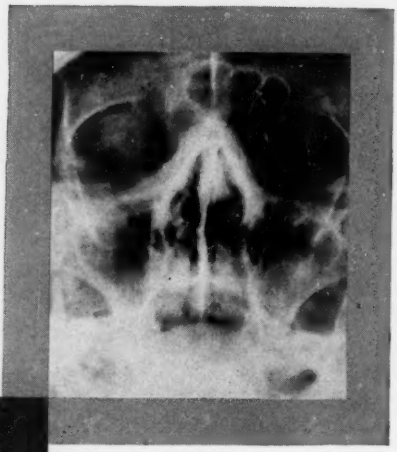

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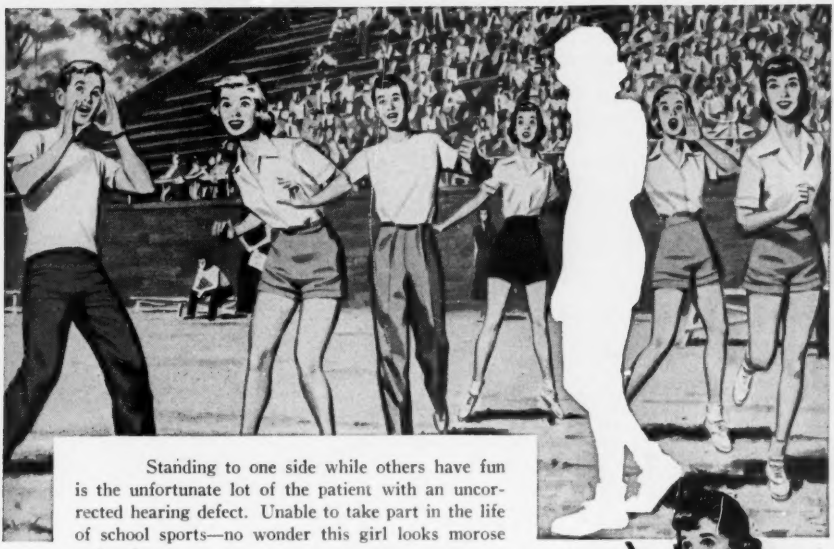

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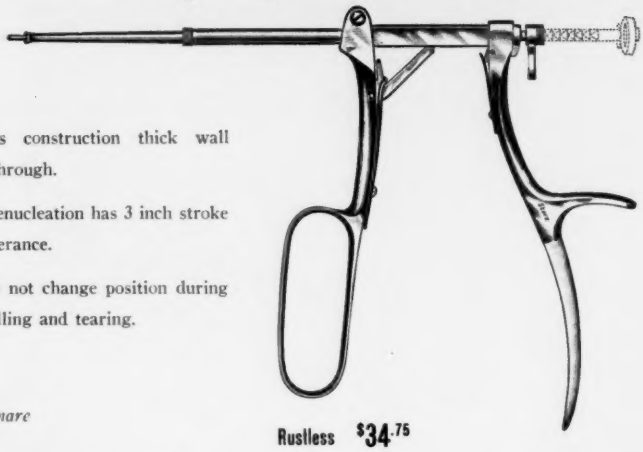

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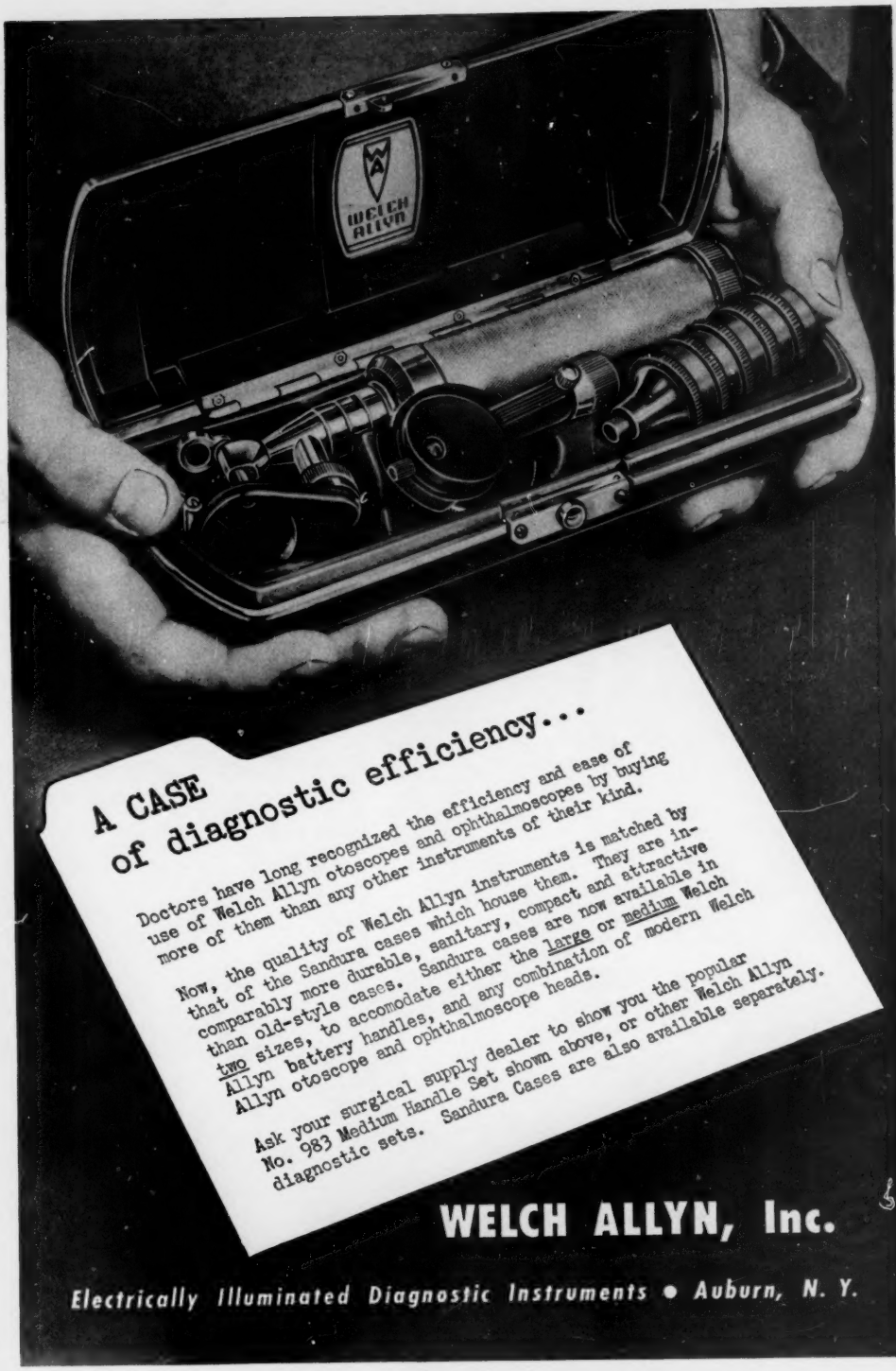

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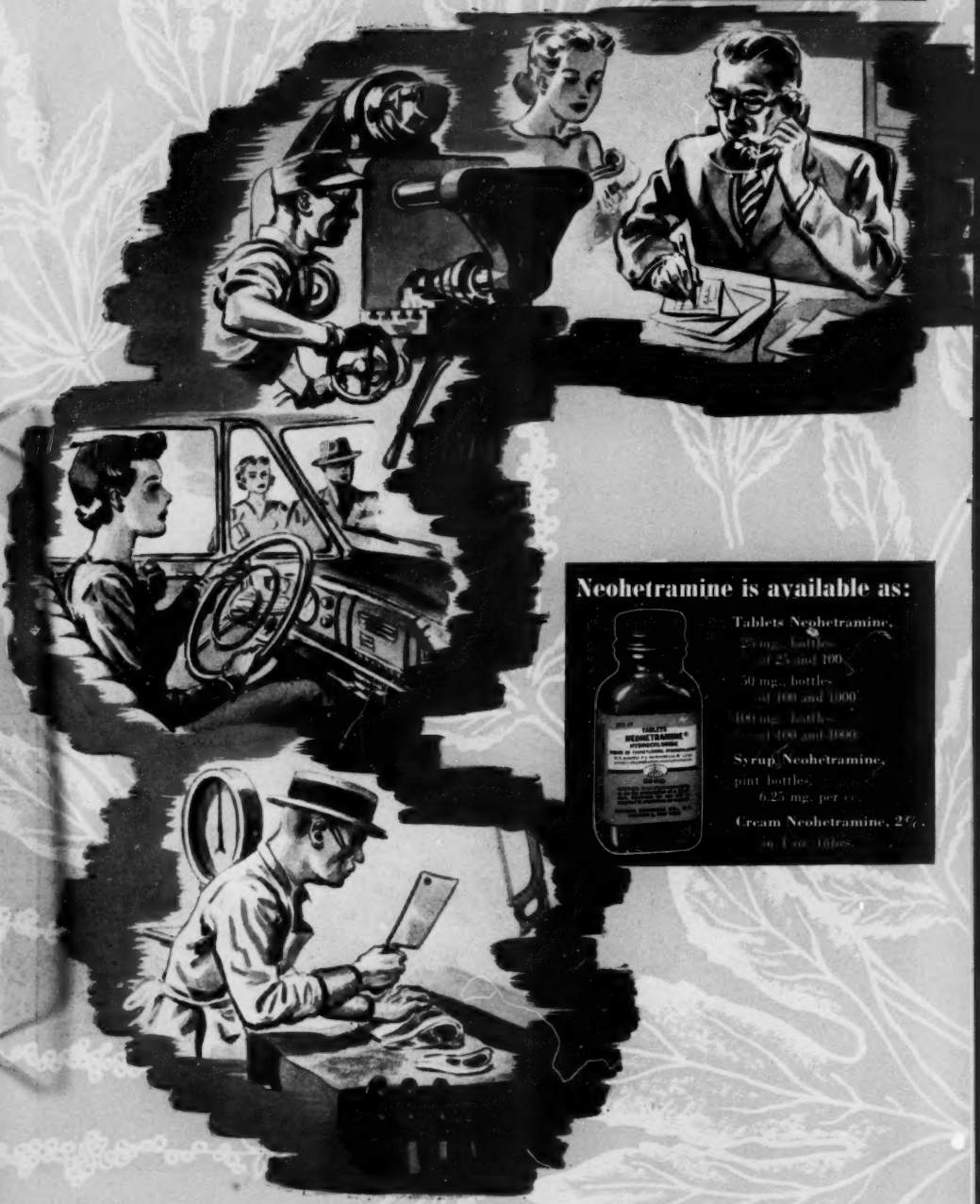
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1. New and Nonofficial Remedies, American Medical Association, Chicago, 1950, P. 29.
2. Friedlaender, S. M., and Friedlaender, A. S.: J. Lab. & Clin. Med. 33:865 (July) 1948.
3. Schwartz, E.: Ann. Allergy 7:770 (Nov.-Dec.) 1949.

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CONTENTS

PAGE

Influence of Fundamental Concepts in Allergy upon Specific Problems in Otolaryngology. FRANCIS L. WEILLE, M.D., AND A. B. RICHARDS, B.S., BOSTON 231

Malformations of the Eustachian Tube, the Middle Ear, and Its Appendages: A Critical Review. FRANZ ALTMANN, M.D., NEW YORK 241

Physical and Physiological Variables in Noise-Induced Hearing Loss. DOUGLAS WHEELER, PH.D., LOS ANGELES 267

Vocal-Cord Paralysis in Heart Disease. MILTON PLOTZ, M.D., AND MORRIS J. BROOKS, M.D., BROOKLYN 273

Prevention of Otitis in the Swimmer. W. VERNON HOSTELLEY, M.D., PHILADELPHIA 279

The Future of Otolaryngology: President's Address. SHERMAN L. SHAPIRO, M.D., CHICAGO 304

CASE REPORTS:

Bilateral Abductor Paralysis Complicated by Fractured Ribs. BERT DEBORD, M.D., TEMPLE, TEXAS 308

King Operation for Cerebral Abscess: Report of a Case Complicating Chronic Mastoiditis, with Cholesteatoma and Labyrinthitis. WALTER D. CHASE, M.D., BETHLEHEM, PA. 309

PROGRESS IN OTOLARYNGOLOGY: Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1950.

Peroral Endoscopy. F. J. PUTNEY, M.D.; J. J. O'KEEFE, M.D.; T. E. DOUGLAS, M.D., AND F. J. MCNELIS, M.D., PHILADELPHIA 312

ABSTRACTS FROM CURRENT LITERATURE 332

BOOK REVIEWS 339

NEWS AND COMMENT 339

DIRECTORY OF OTOLARYNGOLOGICAL SOCIETIES 340

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VOLUME 54 SEPTEMBER 1951 NUMBER 3

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INFLUENCE OF FUNDAMENTAL CONCEPTS IN ALLERGY UPON SPECIFIC PROBLEMS IN OTOLARYNGOLOGY

FRANCIS L. WEILLE, M.D.
AND
A. B. RICHARDS, B.S.
BOSTON

THIS PAPER has a twofold purpose: (1) to review some of the fundamental concepts of the allergist and of the rhinologist regarding sinus surgery in asthma and (2) to report what happened to 197 asthmatic patients who had sinus operations and were followed an average of 8.7 years. The effect on the asthma and on the nose, together with the operative technique, will be discussed.

There is a sharp difference in the management of asthma and vasomotor-rhinitis patients. In the latter disease, if the symptoms are not relieved by the allergist, he or the patient will urgently demand aid from the rhinologist. In the former, the control of wheezing and the suffering entailed is the constant responsibility of the allergist. His opinion as to the merits of any treatment naturally determines what is done. This is good both for the patient and for the otolaryngologist, who as a collaborator must have various skills which often aid in the control of asthma. The judgment of the allergist as to nose and throat problems in asthma is of very great importance because his opinion is either final or causes much procrastination.

One of the most troublesome concepts in the field of allergy is the influence of sinus surgery on asthma in patients having gross pathological changes in one or more sinuses. Cooke and others [1] believe that the long-term effect of such treatment is favorable, while Rackemann [2] has expressed the opposite view. He says: "Operations on the sinuses usually do harm and not good to the asthma" and destroy "the normal structure and function of the nose." Such conflicting opinions would not need emphasis were it not for the fact that lesions of the sinuses in asthmatic patients are very common. The examination of the nose and throat in 500 unselected asthma patients revealed the data shown in Table 1.[3] It is obvious

---

Read before the 36th Clinical Congress of the American College of Surgeons, Boston, Oct. 24, 1950.

From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary and the Allergy Clinic of the Massachusetts General Hospital.

1. Cooke, R. A., and others: Allergy in Theory and Practice, Philadelphia, W. B. Saunders Company, 1947.

2. Rackemann, F. M.: New Concepts of the Causes of Asthma, J. Michigan Med. Soc. **46**:328 (March) 1947.

3. Weille, F. L.: Studies in Athma: The Nose and Throat in 500 Cases of Allergy, New England J. Med. **215**:235-239 (Aug. 6) 1936.

that at least some of the cases in this table sooner or later required an opinion as to what should be done about the nasal disease.

If it is possible to base a decision on the experience of a substantial group of unselected cases observed over a very long follow-up period, clinical judgment founded on facts can be substituted for impressions derived from sporadic successes or failures. Everyone has observed patients who have had less or no asthma after sinus operations as well as improvement in the local nasal condition. Even the most pessimistic allergist would agree to this statement. But the otolaryngologist must consider seriously the allergist's claim [2] that aggravation and not alleviation of the asthma and of the condition of the nose is the usual result, even if this is a fallacy. The allergist can equitably demand that the end results have no adverse effect in the great majority of cases. If he can prove that harm usually occurs, his position is sound in discarding any drastic sinus therapy. In any event, it is he who must approve and more or less permit any sinus surgical venture. If he is inclined toward pessimism, the crux of any decision which he may make resolves itself into three questions:

1. Will the proposed operation usually make the asthma worse?

TABLE 1.—*Nose and Throat Lesions in 500 Unselected Asthma Patients*

Lesion	Total	Extrinsic	Intrinsic	Miscellaneous
Pansinusitis (unilateral or bilateral)	46	11	34	1
Sinusitis—one or more (unilateral or bilateral)	316	136	180	0
Vasomotor rhinitis	223	111	109	3
Polypi	211	81	128	2
Surgically deviated septum	95	47	46	2

2. Will the operation make the condition of the nose worse?

3. What operative techniques are desirable if surgery is undertaken?

In order to provide answers, a group of about 280 asthmatic patients who had had sinus operations were studied. In arriving at an intelligent evaluation of the group, it is first necessary to have in mind a working classification of asthma, with some of its basis in theory and fact. This promotes understanding of how selection of the cases was usually made.

Cooke [1] has classified asthma into the categories of "infective" and "noninfective" or a combination of the two. Rackemann [4] has put asthma patients into two major categories: (1) those presenting a problem in immunology—the extrinsic asthma patients, and (2) others who are a physiological problem—the intrinsic asthma patients.

EXTRINSIC ASTHMA

By this is meant a younger "immunological" group, comprising about 75% of all patients, usually under the age of 30 at the onset of the disease, who ordinarily have

4. (*a*) Rackemann, F. M.: A Working Classification of Asthma, Am. J. Med. **3**:601 (Nov.) 1947; (*b*) Other Factors Besides Allergy in Asthma, J. A. M. A. **142**:534 (Feb. 25) 1950; (*c*) Allergy: A Problem in Immunology; Asthma: A Problem in Physiology, Bull New England M. Center **3**:199 (Aug.) 1941; (*d*) Clinical Allergy, Particularly Asthma and Hay Fever: Mechanism and Treatment, New York, The Macmillan Company, 1931.

1. Sensitivity to one or more foreign proteins demonstrable by a careful history and often confirmed by skin tests.

2. A family history of asthma, hay fever, hives, or eczema.

3. Skin-sensitizing serum antibodies, known as reagins, which are demonstrable by passive transfer (Prausnitz-Küstner reaction) to the skin of normal persons.

4. Eosinophilia in the blood or respiratory secretions, especially nasal.

5. Multiple clinical manifestations of allergy, such as eczema in infancy, hay fever in childhood, asthma in adult life, etc.

This extrinsic group can, in general, be treated by the allergist's methods of desensitization, elimination or "allergic cleanliness," and other medical means. His results,[4d] roughly, are 20% cured, 60% improved, and 20% unimproved. These figures are mentioned merely for orientation of otolaryngologists as to what to expect and for comparison with their own results.

A few practical points call for emphasis. The extrinsic asthma patients just described are sometimes classified as having atopy, i. e., spontaneous presence of serum reagins, a positive family history for allergy, etc. The most important fact for otolaryngologists to understand is that these patients are never cured of their reagin antibodies by any means known today, even though a clinical cure may result in the disappearance of all symptoms. It is possible that desensitization is accomplished by some mechanism such as the post-treatment blocking antibody described by Cooke [5] and Loveless.[6] It may be that treatment really accomplishes its purpose by causing useful greater patient sensitivity. In any event, the allergist's management is basically symptomatic, since reagins are never cured. It is for this reason understandable why, for example, ragweed sensitivity may require seasonal or other treatment for decades or indefinitely.

However, occasionally spontaneous remissions occur in this group so that no treatment of any kind is required for varying lengths of time.

In true extrinsic asthma, the allergist is usually able to satisfy the patient and himself without assistance from the otolaryngologist, even though there may be incidental pathological conditions of the nose and throat. Cooperative effort may be required in cases of "asthmatic bronchitis" in young people. Here, acute respiratory infections cause wheezing even when there has been adequate management of the allergy, and the otolaryngologist, by medical or surgical skills, may lessen the incidence and severity of such acute flare-ups.

Further aid may be given to vasomotor-rhinitis patients who gradually develop asthma (about 18%)—usually severe—despite the allergist. Methods already reported [7] regarding the treatment of vasomotor rhinitis by the rhinologist may have dramatically good effects upon both the nose and the asthma.

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5. (*a*) Cooke, R. A.; Barnard, J. H.; Hebald, S., and Stull, A.: Serological Evidence of Immunity with Coexisting Sensitization in a Type of Human Allergy (Hay Fever), J. Exper. Med. **62**:733 (Dec.) 1935. (*b*) Cooke, R. A.; Loveless, M., and Stull, A.: Studies on Immunity in a Type of Human Allergy (Hay Fever): Serological Response of Non-Sensitive Individuals to Pollen Injections, ibid. **66**:689 (Dec.) 1937.

6. Cooke, Loveless, and Stull.[5b] Loveless, M. H.: Immunological Studies in Pollinosis: I. The Presence of Two Antibodies Related to the Same Pollen-Antigen in the Serum of Treated Hay Fever Patients, J. Immunol. **38**:25 (Jan.) 1940.

7. Weille, F. L.: The Effect of Nasal and Sinus Surgery upon the Manifestations of Allergy, New England J. Med. **242**:43 (Jan. 12), 1950.

Groups of extrinsic cases of particular interest to the otolaryngologist are typified by ragweed-asthma extending into the winter. Respiratory or other infections are supposed to be important in such patients, who are classified as having "extrinsic infective asthma."

INTRINSIC ASTHMA

Intrinsic asthma, usually a problem in pathological physiology, begins ordinarily after the ages 30 to 40 and persists without relation to season, diet, or changes in environment. If it clears up for a while, it may reappear when any severe acute respiratory infection occurs. Skin tests are either flatly negative or not helpful. In some of the patients, bronchial exudate from bronchiectasis or heart disease is important in causing wheezing. Others have emphysema, but many have "vasomotor bronchitis," which is the homologue of vasomotor rhinitis; turgescence of bronchial mucosa and bronchial exudate primarily cause the wheezing. The validity of the classical time-honored idea that bronchiolar-smooth muscle spasm is of greatest etiological importance is doubtful in many cases, judging from bronchoscopic and autopsy evidence.

It is intrinsic asthma and extrinsic asthma complicated by infection which especially demand attention from otolaryngologists and more particularly the problem of surgical treatment of severe chronic sinusitis in these patients. For the past two years, an intensive effort has been made to evaluate exactly our own experience over a 20-year period in this controversial field, particularly the end results. In the group mentioned above of about 280 cases, it has been possible to secure adequate information after a minimum follow-up of six months and a maximum of 26 years in 197 cases, with an average follow-up for eight to nine years. The asthma began before the age of 30 in 79 patients, after 40 in 63 patients, and between 30 and 40 in 55 patients. There was no selection in the group, except for a minimum time of observation and enough facts to justify an opinion as to results in the condition of the nose and asthma. Whenever possible the patient was interviewed and examined directly. He was asked to turn in a detailed questionnaire if he could not be seen for an interview.

This material is mostly from the Massachusetts Eye and Ear Infirmary and Massachusetts General Hospital and will be presented in answer to the questions enumerated at the beginning of this paper. The first two questions will be considered together.

WILL SINUS SURGERY USUALLY MAKE ASTHMA WORSE? WILL IT MAKE THE CONDITION OF THE NOSE WORSE?

In 197 patients having an average follow-up of 8.7 years, 59 were cured or essentially cured of asthma (30 and 29, respectively). Forty-three patients improved. The remainder were the same (59) or worse (36), i. e., 102 were benefited and 95 were not, or 52% and 48%. Intrinsic or unclassified asthmatic patients numbered 131; others classified as extrinsic or with demonstrable allergy not having a proved relation to the asthma equaled 66. The above facts are summarized in Table 2.

It seemed of great importance to determine the results in the asthma and the nose in terms of the time of follow-up. Was the long-term effect good or bad? The fact that the conditions of 84 patients could be reported 10 to 20 years after

surgery and 113 up to 10 years seemed to offer an excellent opportunity to secure valuable information regarding time effects. This information might aid in determining the accuracy of the conflicting claims that while immediate results were often satisfactory, the long-term status would be poor or vice versa.

Therefore, a division of the patients was made into five year periods of observation and the situation regarding the asthma and nose evaluated. That is, 66 patients

TABLE 2.—*Results of Sinus Surgery Compared in Allergic and Nonallergic Asthmatic Patients*

Asthma Result	Asthma Classification				
	Extrinsic	Intrinsic or Unclassified	Totals		Per Cent
Cured	10	20	30		
Essentially cured	10	19	29	102	52
Improved	15	28	43		
Same	24	35	59		
Worse (18%)	7	29	36	95	48
Totals	66	131	197		100

TABLE 3.—*Results in Asthma and Condition of Nose in Sixty-Six Patients Followed Six Months to Five Years After Sinus Surgery*

Results	Asthma			Nose		
	Patients	Per Cent	Per Cent	Patients	Per Cent	Per Cent
Cured	11	16.7		2	3.0	
Essentially cured	9	13.6	50	20	30.3	64
Improved	13	19.7		20	30.3	
Same	23	34.9		20	30.3	
Worse	10	15.2	50	1	1.5	36
Unknown	0	0		3	4.6	
Totals	66	100.1	100	66	100.0	100

TABLE 4.—*Results in Asthma and Condition of Nose in Forty-Seven Cases Followed Five to Ten Years After Sinus Surgery*

Results	Asthma			Nose		
	Patients	Per Cent	Per Cent	Patients	Per Cent	Per Cent
Cured	2	4.3		5	10.6	
Essentially cured	6	12.8	38	3	6.4	53
Improved	10	21.3		17	36.2	
Same	17	36.2		20	42.5	
Worse	12	25.6	62	1	2.1	47
Unknown	0	0		1	2.1	
Totals	47	100.2	100	47	99.9	100

were followed up to five years, 47 patients to 10 years, 39 patients to 15 years, and 45 patients to about 20 years.

Tables 3, 4, 5, and 6 summarize the findings. Table 7 compares the asthma results of the four groups and Table 8 the nasal results.

In regard to the asthma, the figures indicate that, aside from any immediate results under six months (often favorable), half of the patients were improved over a five-year period; 15% were worse. During the second five years, 38% of 47 patients were better; 26% were worse. In 39 patients observed 10 to 15 years, 59% were improved, with 18% worse. In 45 patients followed more than 15 years,

TABLE 5.—*Results in Asthma and Nose Conditions in Thirty-Nine Patients Followed Ten to Fifteen Years After Sinus Surgery*

Results	Asthma			Nose		
	Patients	Per Cent	Per Cent	Patients	Per Cent	Per Cent
Cured	9	23.1		7	18.0	
Essentially cured	7	18.0	59	9	23.1	77
Improved	7	18.0		14	35.9	
Same	9	23.1		6	15.4	
Worse	7	18.0	41	1	2.6	23
Unknown	0	0		2	5.1	
Totals	39	100.2	100	39	100.1	100

TABLE 6.—*Results in Asthma and Nose Conditions in Forty-Five Patients Followed Fifteen or More Years After Sinus Surgery*

Results	Asthma			Nose		
	Patients	Per Cent	Per Cent	Patients	Per Cent	Per Cent
Cured	8	17.8		3	6.7	
Essentially cured	7	15.6	62	14	31.1	78
Improved	13	28.9		18	40.0	
Same	10	22.2		7	15.6	
Worse	7	15.6	38	2	4.5	22
Unknown	0	0		1	2.2	
Totals	45	100.1	100	45	100.1	100

TABLE 7.—*Asthma Results After Sinus Surgery Varied with Time in 197 Patients*

Duration of Follow-Up	Cured	Essentially Cured	Improved	Same	Worse
6 mo. to 5 yr. in 66 patients	16.7%	13.6%	19.7%	34.8%	15.2%
		50%		50%	
5 to 10 yr. in 47 patients	4.3%	12.8%	20.6%	36.4%	25.6%
		38%		62%	
10 to 15 yr. in 39 patients	23.1%	18.0%	18.0%	23.1%	18.0%
		59%		41%	
15 yr. or more in 45 patients	17.8%	15.6%	28.9%	22.2%	15.6%
		62%		38%	

TABLE 8.—*Variation of Results in the Nose with Time After Sinus Surgery in 197 Asthma Patients*

Duration of Follow-Up	Cured	Essentially Cured	Improved	Same	Worse	Unknown
6 mo. to 5 yr. in 66 patients	3.0%	30.3%	30.3%	30.3%	1.5%	4.6%
		64%			36%	
5 to 10 yr. in 47 patients	10.6%	6.4%	36.2%	42.5%	2.1%	2.1%
		53%			47%	
10 to 15 yr. in 39 patients	18.0%	23.1%	35.9%	15.4%	2.6%	5.1%
		77%			23%	
15 yr. or more in 45 patients	6.7%	31.1%	40.0%	15.6%	4.5%	2.2%
		78%			22%	

favorable changes occurred in 62%; 16% were worse. In the 10- to 20-year observation period of 84 asthmatic patients, about 60% had maintained improvement, while approximately 17% were worse.

It is recognized that asthma is a disease characterized by remissions and exacerbations, and that some of the results—whether for better or for worse—are probably unrelated to the sinus surgery. But in this group of unselected cases as a whole, it was not true that sinus operations in asthmatic patients were followed by aggravation in the majority but usually in less than 20%. This is not very different from Rackemann's results in 1,074 patients [4d] treated by all methods. Since the group included many extremely ill patients who had had all manner of other treatment, it is surprising to find so few who were worse. It is of great interest that the long term trend of the asthma was toward improvement more often than not.

This seems to provide substantial evidence regarding fundamental concepts in this specific problem; the allergist who believes that the great majority of

TABLE 9.—*Correlation of Asthma and Nose Results Following Sinus Surgery in 197 Asthmatic Patients*

	Nose Results—Patients					
Asthma Results	Cured	Essentially Cured	Improved	Same	Worse	Unknown
Cured	8	15	4	2	0	1
Essentially cured	2	16	9	2	0	0
Improved	2	8	22	10	0	1
Same	2	5	21	24	2	5
Worse	3	2	13	15	3	0
Total nose results	17	46	69	53	5	7

asthmatic patients who undergo sinus surgery will, in the end, grow worse is in error. So is the otolaryngologist who claims essential "cure" in most cases.

Average improvement in the nose condition occurred more frequently than in the asthma. The local condition was no worse postoperatively in 185 of 190 patients followed an average of 8.7 years. This is evidence against the opinion of both allergists and certain laymen that any type of sinus surgery makes the nose worse.

Table 9 correlates the result in the nose with the status of the asthma when the patient was last seen. Both the asthma and the nose condition were found to be cured, or essentially so, in 41 patients or 21%. This is striking, since a large proportion of the 197 patients had formerly been grouped in the 20% "unimproved" category [4d] of the allergist.

Many paradoxes are apparent. For example, four patients in whom postoperative nasal findings were essentially the same as prior to surgery were cured of asthma, or very nearly so. One example illustrates the difficulties in a presentation of this kind: a 49-yr.-old white woman had intractable intrinsic asthma, vasomotor rhinitis, a gross deviation of the septum, nasal polypi constantly recurring, and bilateral pansinusitis. For 10 years after nasal and sinus surgery, she had no asthma and an excellent nasal result. This apparent "cure" was rudely interrupted when a hysterectomy was done for prolapse of the uterus. The asthma came back after this operation, but the nasal status remained satisfactory.

The fear that both the asthma and local nasal condition may become worse after sinus surgery sometimes causes the allergist to do nothing about gross sinus pathology for years or until the increasing severity of the chest symptoms forces action. He then expects an operation to reverse the situation in the nose and chest. While this hope is frequently unfulfilled, it is important to emphasize that in the great majority of cases there is no adverse change. The single reservation is that reasonable judgment must be used in the selection of the surgical measures to be carried out.

WHAT OPERATIVE TECHNIQUES ARE DESIRABLE IF SURGERY IS UNDERTAKEN?

It is our opinion that intranasal ethmosphenoidectomy, the Caldwell-Luc operation of the antrum, and surgical measures required for good mechanical and physiological functioning of the nasal cavities [7] are adequate in the majority of cases.

TABLE 10.—*Nasal and Sinus Surgery Done on 197 Patients with Asthma*

Asthma Results			Cured	Essentially Cured	Improved	Same	Worse	Totals
	No. of patients		30	29	43	59	36	197
Asthma diagnosis	Intrinsic		18	20	28	40	29	135
	Extrinsic		7	6	8	11	3	35
	Both		1	1	4	5	3	14
	Unclassified		4	2	3	3	1	13
Sinus surgery	Antrum	Int.	16	10	15	19	8	68
		Ext.	12	17	22	32	22	105
	Ethmoid	Int.	14	19	23	36	18	105
		Ext.	2	2	8	7	7	26
	Frontal	Int.	0	0	1	2	1	4
		Ext.	2	1	4	1	3	11
	Sphenoid	Wind.*	2	1	4	3	2	12
		Exen.	1	2	1	1	4	9
	Procedure unknown		0	0	0	2	3	5
Nasal surgery	Polypectomy		13	13	28	36	19	109
	Diathermy cauterization		9	9	9	9	4	40
	Turbinectomy		18	11	19	31	16	95
	Submucous resection		5	10	21	16	12	64

* Windowing.

These procedures in general comprised the surgery in the group herewith reported, except for a radical external approach, typified by the Lynch frontoethmosphenoidectomy, in 11 patients and external ethmoidectomy in 15 others. Intranasal antrostomy was done when, because of other procedures, it was not possible to carry out the Caldwell-Luc technique at that stage or when the antral disease seemed minor.

Table 10 summarizes the various operations and the asthma results. The results obtained in the asthma by external surgery and the comparable intranasal approach are compared in Table 11.

Cases in which both external and intranasal techniques were used are listed in Table 11 as external only. The figures include all possible cases from the 197.

There are four important causes for poor results in the nose: (1) radical surgery of the frontal sinus; (2) recurrent polyposis; (3) failure to control vasomotor rhinitis and mechanical nasal obstruction, and (4) surgery based upon roentgenogram reports almost entirely, the importance of mild sinusitis being

thereby overestimated, especially in patients who have few complaints relating to the nose. The problem of exenteration of the frontal sinus by the Lynch or related methods raises the question of "complete" and "incomplete" removal of pathological sinus tissue in asthma. The usual disease process encountered is polypoid degeneration in one or more sinuses, with or without retained secretion. To remove it completely from the frontal sinus or to reach all anatomical prolongations of ethmoid-cell patterns requires an external frontoethmoidectomy. A previous review [8] of 276 patients of all types having such operations, including 10 of the 11 asthmatic patients mentioned above, shows that secondary surgery must be performed upon the frontal sinus because of complications sooner or later in about one-third of the patients. Because of this fact there must be convincing evidence of necessity—usually a complication, such as an orbital abscess or incipient intracranial disease—before committing the patient to such an operation.

This reasoning does not apply to external ethmoidectomy if the frontal sinus is not entered. However, intranasal ethmoidectomy, if thoroughly done, will remove the pathological tissue adequately. If it is not practical to excise the lining of the

TABLE 11.—*Conservative Versus Radical Surgery in the Ethmoid and Frontal Sinuses: 127 Patients*

Asthma Results	Intranasal		External	
	Patients	Per Cent	Patients	Per Cent
Cured	13	12.7	2	7.7
Essentially cured	19	18.8	2	7.7
Improved	22	21.8	8	30.8
Same	35	34.6	7	26.9
Worse	12	11.9	7	26.9
Total	101	99.8	26	100.0

frontal or the sphenoid sinus completely (in the latter because of danger to neighboring structures), it is of academic interest as to whether or not ethmoid-cell prolongations, as over the orbit, are dealt with.

It is a simple matter to do a bilateral intranasal ethmoidectomy in one stage, and this technique appeals to the patient because no external incisions are necessary. External ethmoidectomy is more time-consuming, especially if bilateral, and one often encounters strenuous objection in female patients because of dread of facial scars, however inconspicuous. The problem of dealing with antral disease is so relatively simple that it will not be discussed here, except to say that the Caldwell-Luc technique was used frequently in the patients (105) reported.

A curious paradox is the failure of both allergists and otolaryngologists to understand the nature of nasal and sinus polyposis. This is unknown, despite the claim [2] that it is "a part of the asthma." If this were so, polypoid recurrence after nasal and sinus surgery, whether very conservative or very radical, could be easily understood. If the allergist accepts such a concept as fundamental, he must urge both the patient and the rhinologist to exercise reasonable care in preventing a poor local result in the nose by destruction of such polypoid tissue as it recurs.

8. Weille, F. L.: The Problem of Secondary Frontal Sinus Surgery, Ann. Otol. Rhin. & Laryng. **55**:372 (June) 1946.

Mechanical and physiological considerations in relation to the control of vasomotor rhinitis and postoperative polypoid degeneration have been previously reported [7] in a group of 272 patients with vasomotor rhinitis treated surgically. The experience therein described applied equally to asthma patients and was employed in their management. The tendency of polypoid tissue to recur and thereby interfere with a good local nasal result cannot be overemphasized. Of equal importance is the ability of uncontrolled vasomotor rhinitis to produce nasal obstruction.

It is rarely desirable to operate upon asthma patients who have minor sinus disease, as shown by roentgenograms, with almost no nasal complaints. If surgery is undertaken, it is important for the local nasal disease to require intervention regardless of the asthma, so that the patient has a reasonable chance for a satisfactory local result, whether or not there is benefit to the asthma.

CONCLUSIONS

A study of 197 asthmatic patients who had undergone sinus surgery and who were followed from six months to 20 years, with an average of 8.7 years, indicates the following:

1. The asthma became worse in 18% of the group and not in 82%. Fifty-two per cent were better.

2. The condition of the nose became worse in five of the 197 patients and not in 185 patients. Two-thirds of the patients were better so far as the nose was concerned.

3. Relatively conservative surgical measures seemed adequate in this group, and were followed by slightly better results in the asthma than when more radical methods were employed.

4. The longer the patients were followed the better were the over-all results, both in the nose condition and in the asthma, as judged by 84 patients followed 10 to 20 years.

Since the judgment of the allergist is usually final in the decision to do sinus surgery in asthmatic patients, he may obtain some reassurance from the foregoing facts.

MALFORMATIONS OF THE EUSTACHIAN TUBE, THE MIDDLE EAR, AND ITS APPENDAGES

A Critical Review

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MALFORMATIONS of the Eustachian tube and the middle ear without simultaneous involvement of either the external or the inner ear or both are rare. The last review of the subject was made by Marx, 1926. Since that time a considerable amount of new material has accumulated, which made another comprehensive study advisable. This seemed particularly worth while in view of the renewed interest in the operative treatment of certain ear malformations.

EUSTACHIAN TUBE

Pharyngeal Ostium.—Although generally the pharyngeal ostium is situated in the fetus below the level of the hard palate, at birth at the level of the hard palate, and in adults about 10 mm. above it, individual variations in the position and shape of the ostium are frequent. Overdevelopment of the tensor tympani muscle may lead to abnormal patency of the ostium (von Kostanecki). Abnormal width of the ostium is not too rare. Sometimes a recessus is formed which extends downward from the ostium proper, between the plica salpingopalatina and the levator ridge. This is the so-called recessus salpingopharyngeus (Zuckerkandl); is separated from the tubal ostium by a mucosal fold. Similar cases have been described by Worms, Coloumna, and van Varseveld. A sickle-shaped membrane, narrowing the ostium from below, has been reported by Mayr.

Diverticula may develop either from Rosenmüller's fossa or from the tube itself. Diverticula which develop from Rosenmüller's fossa extend laterally above the superior margin of the superior pharyngeal constrictor muscle. These so-called Pertik's diverticula are either present at birth or appear later in life (von Kostanecki, Schmidt, Polyák, Körner, Peters, and others). They represent congenital malformations or anomalies and are due to particular anatomical conditions—possibly primary underdevelopment of the tubar anlage. The claim that they originate from a remnant of the dorsal recessus of the second branchial pouch is disputed by Stupka. Among the diverticula of the tube itself the so-called diverticula of Kirchner are most numerous. They originate from the floor near the pharyngeal ostium and expand between the tensor and levator veli palatini muscles. They represent either pulsion diverticula which develop at a typical congenitally weak area

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of the tubal wall (von Kostanecki) or pure malformations (Peters). Tubal diverticula, the so-called air sacs, are regularly present in certain mammals such as Equidae, Tapiridae, Hyracoidea, and Chiroptera (Lechner). Their development starts at a relatively early stage of the embryonic development. It is improbable that the diverticula in man, which are relatively rare, much smaller, and originate from a different part of the tubal wall, are homologous with the air sacs of the mammals mentioned above (Stupka). Occasionally, diverticula are found closer to the middle ear (von Kostanecki, Altmann). In a case described by Cadarso and Goyanes a diverticulum was found below the ostium of the tube, but in contrast to a deep recessus salpingopharyngeus it was separated from the ostium by the levator ridge.

Congenital Tumors.—A pedunculated tumor in a child 8 mo. of age was described by Henke. The tumor originated from the anterior wall of the tube near the pharyngeal ostium and protruded into the middle ear and into the external auditory meatus. It showed the same histological structure as the congenital polyps of the nasopharynx. The tumor was covered with skin with lanugo hairs and sebaceous and sweat glands, and its stroma consisted of connective and fat tissue and striated muscle fibers. Henke also mentioned a few cases from the older literature which were most probably of similar character. Another case has been reported more recently by Schwarzbart.

Sometimes peculiar small polyps are found in the tubal ostium, on the torus tubarius, and in Rosenmüller's fossa (Holmes, Brown, Wood, Tomka, Zöllner). They contain, as a rule, the elements of the pharyngeal mucosa and are regarded as tumor-like hyperplasias on the basis of a faulty anlage (Zöllner).

When severe malformations of the middle ear occur in cases of congenital atresia of the ear, the tube is sometimes normal but often malformed. It is entirely absent, rudimentary, or abnormally narrow. In other instances the cartilage may be absent (Altmann).

DRUM MEMBRANE

The drum membrane is known to have developed not as an independent structure without connection with the neighboring formations but at the place adjacent to the meatal plate and the middle ear. Since the normal formation of the membrane is intimately connected with the normal development of the surrounding parts, malformations of the drum membrane as a whole occur simultaneously with defects in either the inner part of the meatus or the middle ear. These malformations have been dealt with in another paper (Altmann, 1949). Isolated malformations of the drum membrane are extremely rare.

The foramen Rivini, sometimes found at the anterosuperior pole of the membrane, previously considered to be a congenital malformation, is now regarded by most authors as acquired.

Wullstein reports a cyst the size of a pea lined with stratified squamous epithelium which was situated at the border between Shrapnell's membrane and the external canal. He explains its formation by disturbances in the development of the part of the meatal plate which is the anlage of the outer covering of Shrapnell's membrane.

In rare instances small cysts are found within the epidermal layer of the pars tensa of the membrane containing epidermal detritus and cholesterol crystals

(Hinton, Urbantschitsch, Wendt, Küpper, Gruber, McKenzie, and others). These so-called primary cholesteatomas of the drum membrane are comparable to epidermal cysts in other parts of the body and do not represent true malformations.

MIDDLE EAR AND MASTOID

Pneumatization.—When discussing the malformations of the middle ear and its appendages, one should deal first of all with the surprising differences in the structure of the middle ear, the pneumatic cells and their varying topography, the numerous folds of the mucous membrane described as present in the middle ear, and the variations in the external and internal structure of the temporal bone and its parts caused by the above-mentioned variations. Since these do not represent true malformations but physiological or pathological variations, they are not described in this paper. The developmental aspects of the question of pneumatization have already been discussed in another paper (Altmann, 1950).

A peculiar anomaly of the pneumatization of the mastoid was described by Waltner and Karatay. A cyst that had destroyed most of the cortex and formed large defects in the inner table was removed from the mastoid of a young man aged 17, who had a normal drum and no history of previous middle-ear infections. The cyst contained dark, brownish, "lipoidlike" fluid and cholesterol crystals and was lined with cuboid epithelium; its wall consisted of connective tissue with marked round-cell and plasma-cell infiltration. Waltner and Karatay stated that most probably during the first stage of pneumatization of the mastoid process a budding pneumatic cell lined with epithelium might have been detached from the mastoid antrum or the adjoining pneumatic cells. After the separated cell had lost its communication with the main air-containing cavities, cystic fluid accumulated. A similar process in the course of the development of the lungs may result in the formation of an anomaly which is known as congenital cystic lung. Although there is no history of preceding middle-ear infections, they feel that an inflammatory process cannot be ruled out with absolute certainty as a causative factor in the development of the cyst.

A similar case had been reported previously by Amersbach in a woman aged 36. Since she had suffered from a chronic mesotympanic middle-ear infection of the same ear following a scarlet-fever infection in her third year of life, Amersbach assumed that in the course of the infection the antrum had been separated from the mastoid cavity and a "mucocele" had been formed in the latter.

OSSICLES

Malformations of the auditory ossicles in an otherwise normal auditory organ are rare. The stapes is the most frequently affected ossicle. One finds abnormal configuration and insertion of the crura, lack of one or both of them, substitution of both crura by a bony stick, fusion between the crura by a bony plate, overdevelopment of the head, spur-shaped prominences on the crura and the plate, etc. (Tomka, Becker, Hörbst and Sauser). A rare malformation of the stapes of a calf was described by Hyrtl. There was an osseous ankylosis between the posterior part of the footplate and the frame of the oval window; the posterior crus of the stapes originated not from the footplate but from the window frame.

The incus and malleus rarely develop abnormally. An anomaly of the position of the head of the malleus sometimes occurs. In these cases it is situated very close

to the tegmen tympani or lies in a dehiscence of the tegmen just below the dura mater. Later in life persistent mechanical irritation may cause a bony junction between the head of the malleus and the tegmen tympani, the so-called hammer-head ankylosis. Clinically, this may be manifest as a disturbance of sound conduction (Mayer). According to Mayer, a congenital anomaly in the position of the head of the malleus may cause the posterior crus of the stapes to be situated abnormally close to the wall of the niche of the oval window so that constant mechanical irritation may again cause a bony concrescence, with a resulting clinical picture of a disturbance of sound conduction.

This might also have happened in a case reported by Wolff in which an anomalous bony bar extended from the inferior border of the pyramidal eminence to the labyrinthine wall just posterior to the oval window and pressed against the posterior crus of the stapes which showed early signs of pressure erosion. There were no other malformations of the ear. As a rule, however, the formation of bony bridges between the ossicles and the walls of the tympanic cavity due to faulty development is associated with other malformations (Altmann, Nager, Druss and Allen, and others).

Pneumatization of the head of the malformed malleus and of the body of the incus was seen by me in a case of congenital atresia. It has, to my knowledge, never before been described in man and in only some mammals (mole—Bondy, elephant—Kolmer and Eisinger, chimpanzee—Altmann and Vermes).

There are no perfectly reliable observations of supernumerary ossicles in man in otherwise-normal auditory organs.

Malformations of the articulations between the auditory ossicles are extremely rare as solitary malformations. Bony connections between the malleus and incus as well as connective tissue and bony connections between incus and stapes have been observed (Altmann, 1934). These cases can be explained by persistence of the connection between the ossicles in the precartilaginous anlage. A small quadrangular piece of cartilage in the anterior part of the annular ligament in a fetus of 6 mo. of age and in the posterior part of the ligament in an infant of 3½ mo. of age was described by O. Mayer (1917).

An osseous ankylosis between the stapes footplate and the window frame is, according to Hyrtl, not uncommon in Cetacea (whales, porpoises, seals). The observation is not confirmed by Denker and Kernan. Boenninghaus, however, found synchondroses in the place of the incudomalleolar and the incudostapedial joints in whales (Phocaena). The annular ligament of the footplate was almost completely replaced by cartilage in a young specimen. Boenninghaus thought that in older animals the ligament was completely replaced by cartilage and that in many instances the cartilage was replaced by bone; in this way an osseous ankylosis of the footplate would be a common occurrence in old animals. He quoted Hyrtl as saying that such ankylosis does not occur in any other mammal.

Puntigam, however, reported a bilateral ankylosis of the stapes footplate in an ox. According to his description of a macroscopic examination of the specimen, the ankylosis was most probably due to a diffuse calcification or ossification of the annular ligament; there was no evidence of previous inflammations of the middle ear. He felt that ankylosis of the kind described by him was most probably not rare in older animals.

Although microscopic examinations have not been done, it seems quite certain that the above-mentioned cases of stapes ankylosis in certain animals are entirely different in character from the stapes ankylosis in otosclerosis in man in which a circumscribed focus of pathological bone extends into the footplate of the stapes.

The styloid process varies greatly in length, depending upon the extent of downward ossification in the area of the second branchial arch. Abnormal length of the styloid process may cause difficulties in swallowing or neuralgic pains, and a resection of the abnormally long part is sometimes necessitated (von Eicken, Bernfeld, Kabashima, Imai, Fritz, Loeser and Cardwell, Stamm, Eagle, and others).

Severe malformations of the ossicles are frequently observed with simultaneous malformations of the external and middle ear in the group of malformations called atresia auris congenita. In this group all forms of malformations are found: abnormal adhesions, as well as absence of one or more auditory ossicles, and abnormalities of the stylohyal, laterohyal, and tympanohyal bones (Altmann).

MUSCLES

Malformations of the muscles of the middle ear, especially their partial or complete absence, have been observed only associated with other severe malformations. In a few cases a supernumerary muscle has been found (Brock, Altmann).

MUSCLES

Malformations of the muscles of the middle ear, especially their partial or complete absence, have been observed only associated with other severe malformations. In a few cases a supernumerary muscle has been found (Brock, Altmann). This muscle is usually situated in the facial canal, lateral to and above the nerve, and is separated from the latter by a thin layer of bone. As a rule it has no insertion and its ends are free in connective tissue. In one case the terminal sinew adjoined the tendon of the tensor tympani and accompanied it to the manubrium of the malleus. I have stated that this supernumerary muscle is a "rest" of the depressor mandibulae, the opener of the joint of the jaw of the non-mammals, which becomes the joint between the malleus and incus in mammals. Other abnormal supernumerary muscles have been found in conjunction with general malformations of the middle ear.

BLOOD VESSELS

Arteries.—Among the malformations of the arteries, greatest interest is accorded the so-called persisting stapedial artery, which is sometimes considered the cause of endotic murmurs. In the nine cases of the anomaly reported (Hyrtl two cases, Zuckerkandl, Alexander, Lewin, Brock two cases, Adachi, Altmann) the courses of the arteries were practically identical. They branched off from the internal carotid artery, perforated the floor of the tympanic cavity, passed upward over the promontory (in the majority of cases enclosed in an osseous canal of the latter), took their course between the crura of the stapes, entered the facial canal, and finally divided into branches on the outer side of the dura in the area of distribution of the middle meningeal artery. The embryological explanation of the cases of persistent stapedial artery in man seems to be persistence of the conditions found by Fisher in an embryo of 20 mm. Comparative anatomical and embryological investigations

(Tandler, Evans, Congdon, Padget) show that in all mammals, including man, a certain basic pattern exists in the arrangement of the arteries of the head. Among them the stapedial artery plays an important role. It persists completely or partially in a number of species, the rat for example, while in man it usually disappears.

Other arterial anomalies not in the middle ear but in the region of the temporal bone include unilateral or bilateral hypoplasia (Hyrtl, 1836, Guinard, Orr) or complete absence of the carotid artery (Tode, Quain, Peugnet, Wyeth, Wernitz, Flemming, Fisher, Lowrey, Poppi, Hindze and Freidmann, Töndury, da Silva, Wolff), abnormal communications between branches of the external and the intracranial part of the internal carotid artery (Smirnow), anastomoses between the middle part of the basilar artery and the internal carotid artery in the cavernous sinus (about 20 cases), and origin of the vertebral artery from the internal carotid artery near the base of the skull (Gottschau, Batujeff, Oertel, Hirako; details are given by Altmann, 1947). An arteriovenous anastomosis between the internal carotid artery and the venous plexus in the carotid canal was reported by Wittmaack. An anomalous position of the internal carotid artery, unusually close to the tegmen as it traverses the petrous apex, was described by Eggston and Wolff.

Anomalies of the middle meningeal artery are rare. In about 1% of the cases the artery enters the middle fossa through the foramen ovale but shows normal ramification. In these instances the foramen spinosum either is absent or communicates with the foramen ovale (Chandler and Derezinski). In other cases, the anterior branch enters from the orbit and the posterior branch through the foramen spinosum (Chandler and Derezinski, Toida) or both branches enter from the orbit (Toida, Low). In the latter case the middle meningeal artery must have originated from the sphenomaxillary portion of the internal maxillary artery, entered the orbit through the inferior orbital fissure, and left through the superior fissure of the foramen meningo-orbitale in the greater wing of the sphenoid.

In severe malformations of the ear other abnormalities of the arteries interesting from the developmental point of view have been found, but these are more difficult to explain; e. g., the origin of the middle meningeal artery from the occipital artery, the common origin of the middle meningeal and the internal auditory artery from the basilar artery (Altmann, 1932), or the origin of the superficial petrosal branch from the internal auditory artery in a case with anomalous course of the middle meningeal artery (Altmann, 1949).

Mention must be made in this connection of the small artery which is regularly found in the stapedial region (Hyrtl, 1845, Zuckerkandl, Adachi). I believe that this artery originates from the superficial petrosal branch of the middle meningeal artery, accompanies the facial nerve downward, anastomoses with the stylomastoid branch of the posterior auricular artery, and passes downward through a dehiscence in the inferior wall of the facial canal. It is then located in the free margin of a semilunar fold which runs from the posterior wall of the niche of the oval window forward and laterally. While in this fold, the artery passes through the crura of the stapes to the arterial plexus on the surface of the promontory. Considering its origin and course, this vessel most probably represents a rudiment of the middle part of the main stem of the stapedial artery between the stapes and the point of origin of the supraorbital branch. According to Nabeya, the vessel in question is a branch of the stylomastoid artery which anastomoses with the superficial petrosal branch.

Veins.—In the area of the auditory organ, as elsewhere, there is a far greater variability in the course of veins than of arteries, a fact which is easily explained by consideration of the development of the veins. The great clinical importance of individual variations of these veins is demonstrated when one recalls the significance of individual variations in the formation of anastomoses between the superior sagittal and the transverse sinus and the vena cerebri media in the passage of inflammatory processes from the ear to the endocranium (Trolard, Henle, Streit, von Riccabona, and others).

The lateral and sigmoid sinuses have a separate embryologic origin. The former, which takes a horizontal course, is derived from the end part of the anterior and middle plexus; the latter, which takes a vertical course, is derived from the communication between middle and posterior plexus. In accordance with these facts, variations in the two sinuses are independent of each other. The sigmoid sinus shows greater constancy and fewer variations than does the lateral sinus (Waltner).

Slight variations in the course of the sigmoid sinus are of great clinical importance and were dealt with in the consideration of pneumatization of the mastoid process. A marked forward position of the sigmoid sinus is usually associated with a markedly or completely arrested pneumatization of the mastoid. The anteposition is more pronounced, as a rule, on the right side (Okada, Wittmaack, Jemmi, Seppälä, and others). Differences in the width of the sigmoid sinuses and the size of the jugular foramens are not uncommon. The right sigmoid sinus is, as a rule, wider than the left one. Körner found greater width of the right in 77%, of the left in 13% and equal width of both sinuses in 10% of the cases. A markedly contracted jugular foramen with a small jugular vein was found by Linser in 30 of 1,022 skulls examined. It was five times more frequent on the left side. Swift reported a case in which both jugular foramens were rudimentary in a child 3 days old.

High position of the bulb is commoner on the right side. It is often associated with dehiscences in the bony floor of the middle ear (Streit) which might extend into the external auditory meatus (Kono). An extremely high position of the jugular bulb which extended medially from the cochlea to the middle and posterior fossa was reported by Zaufal, Langenbeck, and Specht.

The following malformations of the lateral and sigmoid sinuses are of interest.

1. Absence or marked hypoplasia of the lateral sinus is found with the superior petrosal sinus opening into the sigmoid sinus (Lieutaud, Hallett, Furstenberg, Loebell, von Riccabona).

2. Absence of the sigmoid sinus occurs with the lateral sinus narrow and connecting with the superior petrosal sinus and the inferior petrosal sinus wide (Waltner).

3. Absence or hypoplasia (Hofmann) of the greater part of the sigmoid sinus is found below the level of the emissary vein. The lateral sinus is normal or narrow, leaving through the mastoid foramen; the superior petrosal sinus is normal or wide and the petrosquamous sinus often persistent (Malacarne, Barkow, von Luschka, Zuckerkandl, Henle, Knott, Budde, Buhe, Streit, Haymann, Law, Kraus and Wirkner, Laff, Williams, Hoople, Williams and Hallberg, Kaulich, Loebell).

4. Absence of the lower third of the sigmoid sinus is found with the sinus ending downward in a blind pouch and draining through a large mastoid foramen and the petrosquamous sinus persistent (Furstenberg).

5. Duplications of the lateral and sigmoid sinuses occur, the two channels separated from each other by a bony bridge or fibrous septum (Meckel, Hallett, Knott, Dumont, Streit, Beyer, Hahn, Brown, Kraus and Wirkner, and others).

6. Hernia-like bulging of the outer wall of the sinus, especially in the region of the upper knee, is relatively frequent (Bezold, Zuckerkandl, Hartmann, Brühl, Lüders, Alexander, Ruttin, Kraus and Wirkner, and others).

7. Persistent petrosquamous sinus is relatively frequent (Zuckerkandl, Knott, Cheatle, Streit, Vernieuwe, Laff, and others). After leaving the lateral sinus it usually crosses the ridge of the petrous bone, follows the course of the petrosquamous fissure, and leaves the middle fossa either through the spurious jugular foramen or with the middle meningeal vein through the foramen spinosum or, in rare instances, through the mastoid foramen. In a malformed temporal bone with absence of the foramen spinosum, the sinus left through the foramen ovale (Altmann, 1949). Veins of the middle ear may drain directly into the petrosquamous sinus through the petrosquamous fissure (Cheatle).

8. The terminal part of the sigmoid sinus, just above the jugular bulb, is sometimes covered on its inner surface by bony plates and thus runs through an almost completely closed bony canal (Streit).

9. The sigmoid or lateral sinus is covered by a very thin layer of cortical bone or lies directly under the periosteum (Streit, Kraus and Wirkner, and others) or even in the soft tissues (Furstenberg).

10. The inferior petrosal sinus drains into the superior thyroid vein (Theile).

11. Abnormal width as well as absence of the emissary vein and also presence of several veins are relatively frequent (Streit). Otto observed the vein going into the petrosquamous sinus.

12. Anomalies in the course of the superior petrosal sinus are not uncommon. Instead of going along the superior border of the petrous pyramid directly into the cavernous sinus, it might take a slightly lateral course and run toward the cavernous sinus in either the superior or the inferior wall of the cavum Mecklii (Baldenweck, Altmann, 1933, Case 5). In other cases, the sinus when approaching the region of the gasserian ganglion becomes divided into two branches which reach the cavernous sinus on both sides of the ganglion. Complete absence of the superior petrosal sinus has been reported by Hyrtl and Knott. In a case reported by Furstenberg, it went to the foramen spinosum.

13. Sometimes a so-called ophthalmopetrosal sinus of Hyrtl (accessory sinus of Kelck, aberrant vein of Verga) is present. It connects the ophthalmic vein with the transverse or the superior petrosal sinus or with the venous plexus of the facial canal, thereby entering through the hiatus, or with both venous systems. It is a persistence of a collateral offshoot from the anterior cardinal veins which lie medially to the cranial nerve roots (Stewart).

In addition, the following anomalies are mentioned.

14. Petro-occipital sinus (sinus petro-occipitalis inferior, Trolard, after Knott) is probably a derivative of the posterior cerebral plexus. It runs extracranially

along the inferior surface of the petro-occipital suture. Anteriorly, it traverses an opening in this suture near the apex of the petrous bone and becomes continuous with the carotid venous plexus at the point at which the latter enters the cavernous sinus proper. Posteriorly, this sinus drains into the jugular bulb, where it is in close relation with the inferior petrosal sinus. It receives veins from the pharynx and emissaries from the cavernous blood sinus (Stewart).

15. Persistence of the vena capitis lateralis has been found associated with severe malformations in rare cases (Hofmann, 1931, Hagens, 1932, Altmann, 1933).

NERVES

True malformations of the nerves, with the exception of small and unimportant variations in the course of the chorda tympani or the superficial petrosal nerves without other malformations are not reported. An abnormal course or complete absence of the two last-mentioned nerves or an abnormal course and sometimes a striking undervelopment, a hypoplasia of the facial nerve, may be found in conjunction with more extensive malformations. This may be the reason, or at least one of the reasons, for the congenital palsy of the facial nerve sometimes observed in cases of severe malformations of the ear.

Among the anomalies of the facial nerve in cases of congenital atresia of the external canal, one is of particular interest (Altmann, 1933, Case 1). In this case the hypoplastic facial nerve went through the stapes between the crura in approximately the same way as the stapedial artery sometimes does. Such a course has never been described in man or any mammal. It can be explained only by the assumption that the nerve was from the very beginning abnormally located within the mesenchyme in which the differentiation of the stapedial ring later took place.

Dr. E. P. Fowler Jr. has informed me that he has seen three cases of anomaly of the facial nerve in otherwise normal ears. In the first case, a case of Dr. Schattner's, the facial nerve was normal in its diagonal course across the middle ear but on reaching a point just below the middle of the horizontal semicircular canal it followed the cortex of the external auditory canal wall and came out at the junction of the anterior and inferior portion of the wall instead of descending directly to the stylomastoid foramen. In the second case, a case of Dr. Pierce, part of the facial nerve descended directly in the usual course, but another part, that which innervated the lower part of the face, swung backward from the usual midpoint in the horizontal canal and did not descend until reaching the sinus wall, which was perhaps ½ in. behind the posterior canal wall. On the top of the digastric groove it swung forward again and joined the normal filament just above the stylomastoid foramen. In the third case, the diagonal course of the facial nerve in the middle ear was shortened. However, after making the first bend below the anterior border of the horizontal canal the nerve did not descend directly but followed a more oblique course than usual. The nerve was therefore about ⅜ in. behind the posterior canal at the midpoint of its descent and almost ½ in. behind the posterior canal wall as it left the mastoid through a foramen which was practically in the middle of the posterior part of the mastoid tip.

Cases in which the descending part of the facial nerve took a much more superficial course than usual have been reported by Schwartze, Streit, Yates, Barnhill, and Mellinger, and others (*Flachverlauf des Nerven*, Schwartze).

Mention should also be made of the small deposits of tissue resembling the carotid body which are occasionally found in the adventitia of the dome of the jugular bulb, in the canal that transmits the ramus tympanicus of the ninth nerve through the floor of the middle ear and along the course of this nerve over the promontory (Guild, Lattes and Waltner). Their structure is rather similar to that of the carotid body. They represent parts of a system of masses of nonchromaffine tissue closely associated with the parasympathetic division of the autonomic nervous system and might give rise to tumors, the so-called nonchromaffine paragangliomas in the jugular fossa and the middle ear.

DIFFERENCES IN THE POSITION OF THE PETROUS PYRAMIDS

The petrous pyramids assume different positions in different types of skulls. Of clinical interest are the variations of position found in some asymmetrical skulls. One may find that the pyramid on one side is rotated on its longitudinal axis and in this way there is a shifting of the upper surface of the pyramid—the surface turning upward and approaching the horizontal plane, while the posterior surface becomes more vertical. This, of course, is associated with a change in the position of the tegmen as well as other landmarks of importance.

ABNORMALITIES OF THE BONY WALLS

The most important clinical variations in the bony walls of the middle ear, such as a low position of the tegmen or high position of the bulbus, have already been mentioned. Of clinical importance are dehiscences, occurring in the bony facial canal (Sendulski). A rapid progression of a middle-ear inflammation may occur in this area. Acute inflammations of the middle ear are, therefore, sometimes accompanied by a more or less complete facial palsy which appears during the first days of the infection. This can be explained by a collateral edema of the nerve in such a dehiscence. The palsy may regress spontaneously after a few days. Dehiscences, as a rule, are situated above the niche of the oval window, have an oval shape themselves, and are closed by a connective-tissue membrane. Their occurrence is explained by the fact that the facial nerve originally lies in a sulcus of the labyrinthine capsule which is closed to form a canal by the development of a bony cover on a connective-tissue base in late embryonic life and the first year of postembryonic life. A dehiscence above the oval window niche often occurs in connection with a hyperplastic condition of the mucosa of the middle ear (Wittmaack). The lowest portion of the facial canal is not formed in the above manner but originates, together with certain parts of the posterior and inferior walls of the tympanic cavity and the sheath of the styloid process, from the very posterior parts of the skeletal portion of the second branchial arch, the cartilaginous laterohyal, the later bony tympanohyal (Broman, van Kampen).

In the portion of the tegmen arising from connective tissue, congenital dehiscences are also observed (Sendulski), and although their importance in the passage of inflammatory processes from the middle ear to the endocranium cannot be denied they are insignificant when compared with dehiscences of a different kind whose importance has been studied particularly by Wittmaack (1918, 1926). In newborn infants and during the first few months of infancy, circulatory connections between

dura periosteum and the neighboring portions of the temporal bone, as well as between the mucous membrane of the floor of the tympanic cavity and the bulb of the jugular vein, are very extensive. Therefore, in certain stages of development there are abundant communications between vessels entering the tympanic cavity from the dura and the floor of the tympanic cavity and the vessels of the subepithelial tissue of the middle-ear mucosa. While there is normally a disappearance of the vessels together with the regression of the subepithelial layers of the middle-ear mucosa in cases of persistence of the high subepithelial mucosa, these circulatory connections may be preserved. Consequently, an anatomic condition which enables the conduction of inflammatory processes along these preformed vessels is established for life. Such abnormal vessels are limited in later life to the same areas in which they are found during embryonic development and infancy. The most important of these communications lies in the roof of the tympanic cavity near the point of departure of the superficial petrosal nerves; two others are found in the posterior portion of the pyramid in the neighborhood of the sigmoid sinus, one of them immediately beneath the pyramidal ridge, one near the upper knee of the sinus, and one on the floor of the tympanic cavity near the jugular bulb.

Sometimes there is an insufficient union of the parts which compose the temporal bone, a partial persistence of the open sutures. These are primarily the open petrosquamous or squamomastoid suture, or sometimes the petrotympanic suture. A persisting squamomastoid suture is rare (Frey, Brühl, Körner, Wolff, Kulenkampff). It could be a predisposing factor in the progression of a mastoidal infection toward the outside, as in the development of a subperiosteal abscess. In other instances large parts of the suture were found to be open (Aeby, Wildermuth, Symington, and others). Persistence of remnants of the sutures in adults is, however, by no means rare and has been noticed by Kirchner, Kiesselbach, Adermann, and others.

Complete absence of the inferior wall of the carotid canal in the petrous apex or dehiscences in the latter are due to nonformation or incomplete fusion of the two bony lamellas, which in the course of the normal embryonic development transform the carotid groove into a canal (Kecht).

Complete bilateral absence of the zygomatic process has been reported by Lockhart in a case of, in addition, anomalies of the external and middle ear. In the case reported by Holmes, the zygomatic process was replaced by a fibrous band. An abnormal suture line at the root of the zygoma, between the latter and the squama, was found by Zuckerkandl and Adachi. In Zuckerkandl's case of a 14-yr.-old boy, the squama was hypoplastic. In its anterior part it was incompletely divided into a medial portion which was continuous with the main part and an external or lateral portion which was connected with the underlying medial portion. There was another isolated piece of bone in the posterior part of the mandibular fossa.

An anomaly of the squama of great morphological interest has been described by me in cases of congenital atresia (Altmann, 1933 and 1949). The squama was either normal or hypoplastic, and a part of the greater wing of the sphenoid extended for varying distances backward along the inner surface of the squama. In this area, the lateral wall of the skull was actually formed by two different bones which were separated from each other by a layer of connective tissue. The explanation of the anomaly is difficult. Comparative anatomical studies show that the lateral part

of the middle cranial fossa in mammals forms a region which in lower vertebrates is outside the cranial cavity. Since with the marked increase in size of the brain in mammals the original lateral wall of the cranial cavity has disappeared, the brain was extended laterally into an area which originally was outside of the primordial cranial cavity. A secondary lateral wall of the cranial cavity was formed by the greater wing of the sphenoid (details are given by Matthes and Stadtmüller).

In lower vertebrates the squamosum is a membranous (dermal) bone of the otic capsule and the palatoquadratum. In mammals it retains its relation to the otic capsule. It becomes intercalated between the parietal, petrosum, and greater wing of the sphenoid. Its part in the formation of the lateral wall of the skull shows great variations, however, in different species of mammals. It is, for example, almost completely excluded from the lateral wall in Ruminantia and Cetacea, but in other orders it takes an important part in the formation of the wall. It seems not improbable that the anomaly in question reflects the fact that the role of the squamosum in the formation of the lateral wall of the skull is not a primitive condition but a peculiar characteristic of the Mammalia which shows a great variability in different species.

In this connection the ear changes in certain congenital malformations of the skull will be discussed briefly.

Dyscephaly (oxycephaly, turricephaly, or tower skull) is a congenital deformity of the skull due to hypoplasia of the base and, in most instances, premature synostoses of certain cranial sutures (coronary or sagittal or coronary and lambda sutures). It has been assumed by Nager and de Reynier that it is due to a selective disturbance of the head organizer caused by either hereditary or acquired factors. The findings in the ears vary; sometimes they are free from anomalies. The auricles occasionally show deformities (Haase). Because of the hypoplasia of the base, the external openings of the auditory meatuses are displaced caudally (Günther). The external meatus itself, in some cases, shows more or less extensive hyperostoses (Brunner, Gerlings, and others). The drum membrane often shows scars after middle-ear infections. This is explained by the fact that in dyscephaly one often finds a narrow nasopharynx, large adenoids, and septal deflections (Brunner, Nager and de Reynier). The following histological observations in temporal bones have been reported (Brunner, Gerlings): asymmetry of the temporal bones, hyperostosis of the posterosuperior wall of the external auditory meatus, hyperostoses of the wall of the middle ear, reduced pneumatization, narrowing and elongation of the internal auditory meatus, and marked sharpening and prominence of the petrosal ridge. The inner-ear capsule remained normal. The organ of Corti was atrophic and the spiral and vestibular ganglia partially atrophic in the case reported by Gerlings.

The functional examination of the ear frequently shows, according to Brunner, Ruttin, Nager and de Reynier, and others, disturbances of the cochlear or of the cochlear and vestibular apparatus, often combined with an affection of the optic nerve. The disturbed function of the ear could be due to increased intracranial pressure which usually becomes manifest during the third to the fifth decade and consists of perception deafness, vestibular hyperirritability or hypoirritability, or partial loss of the vestibular irritability (caloric or rotational). The hyperirritability could also be caused by pressure of hyperostoses upon the cerebellum, the reduced

irritability by pressure upon the vestibular nerve (Ruttin). According to Gerlings the auditory as well as the vestibular disturbances would be explained by a stretching of the eighth nerve owing to the elongation of the internal auditory meatus.

The rare acrocephalosyndactyly (Apert) shows a combination of syncephaly with syndactyly on one to four extremities. The auricle was, in some cases, slightly deformed, but there are only insufficient functional and no anatomical examinations of the ears in such cases (details are given by Nager and de Reynier).

Dysostosis cleidocranialis is a congenital, often familial, malformation of the skeleton with dominant hereditary trait. Complete or partial absence of the clavicles is combined with anomalies of the skull (turricephaly with obvious brachycephaly, impeded growth of the membranous bones, and hypoplasia of the facial skeleton) and of the pelvis (defect of the symphysis). Occasionally, additional anomalies are found (Hultkrantz, Antonini). The ear is rarely or never (Delmas-Marsalet) affected. Hultkrantz found marked hypoplasia of the mastoids and Russo-Frattasi saw blue discoloration of the drum membrane on one side which he attributes to a defective ossification of the roof of the jugular fossa. Nager and de Reynier found in three cases a concentric narrowing of the external auditory meatus and hypoplasia of the mastoids, mixed deafness which increased with age, and normal vestibular responses. There was no histological examination of the temporal bones.

Hereditary craniofacial dysostosis (Crouzon's disease) is a rare condition affecting the bones of the cranium (dyscephaly with brachycephaly) and of the face. It may be combined with syndactyly ("cephalodactyly"). In these conditions one frequently finds, according to Nager and Nager and de Reynier, malformations of the external auditory meatus, either marked narrowing of the lumen or defect of the innermost part with absence of the drum membrane. Deformities of the auricle are rare. Malformations of the middle ear with conduction deafness may also be present. The functional examination of the ears with tuning forks (Aubry, Nager and de Reynier, and others) shows reduced hearing of varying degree with lengthened bone conduction and decreased irritability of the vestibular apparatus. The histological examination of two temporal bones by Nager showed hypoplasia of the parts consisting of periosteal (membranous) bone, such as the periosteal labyrinthine capsule and tegmen, with normal development of the parts composed of enchondral bone. The mastoids were hypoplastic. The external auditory meatus was narrow, and in one case the innermost part of the canal was missing. The ossicles were malformed. In one case an abnormal bony bridge extended from the promontory to the capitulum of the stapes; in another case, from the lateral wall of the tympanic cavity to the handle of the malleus. The inner ear was normal, but in one case the septum between middle and upper cochlear turn had remained membranous.

Dysostosis mandibulofacialis (Franceschetti and Zwahlen) comprises malformations of the eyelids, hypoplasia of the zygoma and the mandible, microtic with extreme narrowness or atresia of the external auditory meatus, frequently macrostomia and preauricular fistulas, and anomalies of the teeth, hair, and hard palate. The functional examination shows conduction deafness. No histological examination of the temporal bones has been made (details are given by Franceschetti and Klein).

Dysostosis mandibularis (Nager and de Reynier) is a hypoplasia of the mandible with anomalies of the teeth and with microtia and congenital atresia of the external auditory meatus. It can be regarded as the mildest degree of otocephaly. No histological examination of the temporal bones has been made.

DERMOIDS AND CHOLESTEATOMAS

In discussing the malformations of the middle ear, the dermoids and the true cholesteatomas, heterotopic new formations on the basis of congenitally displaced epidermal elements, must be mentioned.

In the middle ear or mastoid, dermoids containing epidermal lamellas, hairs, and other adnexa of the skin have been described by Toynbee, Hinton, Wagenhäuser, Scheibe, Ribbert, Urbantschitsch, Grünwald, Pavlica, Watanabe, Portmann and Corgnet, and others. A dermoid of the temporal fossa has been reported by Czurda.

Congenital cholesteatomas are found in various bones of the skull, such as the frontal, parietal, sphenoid, occipital, and temporal bones. They originate from displaced epidermal (ectodermal) elements, either within the bone itself or on the outer surface of the dura mater.

Cholesteatomas arising from the region of the temporal squama have been reported by Virchow, Schwartze, Beyer, and Laszkiewicz. Unterberger's and Matschnig's cases most probably belong to this group. Other cholesteatomas arising from the outer surface of the dura of the middle or posterior cranial fossa have eroded the bone on the posterior surface of the petrous bone or the inner cortex of the mastoid (Mondschein, Amersbach, Berger, von Riccabona). The tumors sometimes attain considerable size and bulge outward underneath the periosteum or epidurally toward the cranial cavity. Sometimes similar tumors are found within the cranial cavity, at the base of the brain, in the cerebellopontine angle, in the suprasellar region, and in other locations. They arise from displaced epidermal elements within the leptomeninges or, in rare instances, in the choroid plexus of the third or fourth ventricle and invade the brain secondarily by means of expansive growth.

Two other groups of cholesteatomas are also undoubtedly congenital, cholesteatomas of the middle ear and mastoid in cases of congenital atresia of the external canal (Lüders, Gumperz, de la Bernardie, Goufas, David-Galatz, and possibly also Wanamaker) and the cholesteatomas which are confined to the petrosal tip (Lehmann, Grahe, Beck, possibly also Jefferson and Smalley).

Similar in nature are cysts lined with epidermis, which in rare instances are found in the mastoid (Weaver). Possibly the cavity found by Kelemen in an ear with congenital atresia of the external auditory meatus belongs in this category.

In cases in which the cholesteatomas have extended into the middle-ear spaces, which is apt to happen when the tumors grow, the differentiation between congenital and acquired cholesteatomas becomes very difficult, sometimes even practically impossible. The histological picture of congenital and acquired cholesteatomas is identical. Nevertheless, 20 cases were selected from the literature by Teed in which, in his opinion, the cholesteatoma most probably was congenital, and a few additional observations have been reported since then. In all these cases no ear symptoms other than deafness, tinnitus, or otorrhea has been present, the last for not more than five months prior to the operation and usually for only two to three weeks. In some of the cases, there was no perforation in the drum membrane. The cases of supposedly congenital cholesteatoma are not especially enumerated, since in my opinion in the majority of them the congenital nature has not been proved beyond doubt (bibliography).

Contrary to the more or less generally accepted opinion that congenital cholesteatomas of the ear arise from displaced epidermal cells, it is believed by Teed that they can be derived from the epibranchial placode of the first branchial cleft. He has frequently found in human embryos of about 4 mo. a thickening of the epithelial lining at the dorsal pole of the middle-ear cavity, which in his opinion represents the last vestige of the epibranchial placode. The cells in this area appear similar to those of the basal layer of the meatal plate. Under normal conditions, the cells gradually become transformed into a single layer of flat cells similar to the entodermal lining of the tympanum, so that by the time of birth no differentiation between the two types of cells can be made. Sometimes, however, these ectodermal cells develop in a true island of epidermis. This was observed by Teed in a 5½ mo. fetus. He also referred to a case reported by Přecechtěl, of a newborn child with malformation of the external canal in which islands of epidermis were seen not only in the epitympanum but also in other parts of the middle ear. Islands of stratified squamous epithelium with a tendency to keratinization in the anterolateral part of the tympanum were observed by Mündnich in an infant of 10 wk. He regarded these islands as displaced ectodermal elements; the displacement must have taken place, in his opinion, either very early at the time of the gastrula formation or later during the formation of the middle ear from the ectodermal part of the obturating membrane of the first branchial cleft. Wolff saw multiple patches of squamous epithelium at the aural end of both Eustachian tubes in an infant with cleft palate.

Teed's theory is not sufficiently supported by morphological evidence from actual observations of early embryonic stages. It is hard to understand why congenital cholesteatomas of the middle ear should have an origin so entirely different from that of congenital cholesteatomas in other sites.

Some authors such as McKenzie, Holmes, Diamant, and others consider the possibility that not only the "true" cholesteatomas just described but also many of the ordinary cholesteatomas of the middle ear, particularly the isolated attic cholesteatomas with intact pars tensa of the drum membrane, the so-called genuine cholesteatomas of Wittmaack, might be derived from displaced ectodermal cells. It is, however, difficult to furnish histological proof for this assumption. One would expect to find such cholesteatoma "germs" relatively frequently in the mucosa of the attic. That this is not the case might be owing to the fact that, theoretically, they could be represented by single flat epithelial cells which may escape detection even in a thoroughgoing histological examination (Diamant).

An interesting explanation of the pathogenesis of the cholesteatomas which develop insidiously from a small posterosuperior marginal perforation in the pars tensa has been given by Eigler. He assumed that two pneumatization principles are discernible in the ear, one entodermal in nature which gives rise to the pneumatic cells of the middle ear and mastoid and one ectodermal in nature which normally produces only the lumen in the meatal plate, the epithelial lining of the later osseous external auditory meatus. This epithelium, however, retains its pneumatizing power in a latent stage throughout life, particularly in the posterosuperior part of the meatus, which always remains a center of growth activity.

In cases of impeded entodermal pneumatization the latent ectodermal pneumatizing potency might again become manifest; the epidermis of the posterosuperior

meatal wall might grow through the intact drum membrane into the underlying connective tissue of the middle ear and produce epidermis-lined spaces around the labyrinth which could be regarded as ectodermal pneumatic spaces. This cavity formation is followed by the development of a small marginal posterosuperior perforation and, if the superficial epidermal layers which are desquamated into the lumen of the spaces cannot be emptied into the external auditory meatus, by the formation of cholesteatomas.

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APPENDIX

Cases of Possible Primary Cholesteatomas of the Mastoid

Bernheimer, L. B.: Primary Cholesteatoma of the Mastoid, Ann. Otol. Rhin. & Laryng. **46**:453, 1937.

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PHYSICAL AND PHYSIOLOGICAL VARIABLES IN NOISE-INDUCED HEARING LOSS

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LOS ANGELES

IN THE present state of our knowledge about noice-induced hearing loss, there are two factors as yet imperfectly understood. These are (1) the onset of loss and (2) the rate of progression of loss. Onset is meant here to be the incidence of loss in a known population or sample of a population when the exposure variables are known; rate of progress refers to the rapidity of increase in loss after it has been detected in the person under known exposure conditions.

It is difficult to elucidate these factors directly from the many fine studies which have been conducted on noise and its effects upon hearing acuity. As a rule, the exposure conditions described have been insufficiently quantified. In addition, the losses disclosed have often been assigned to noise by implication, i. e., other etiologies have not necessarily been excluded, either as primary or as contributing causes. We are, therefore, in possession of excellent evidence demonstrating that groups of people working under high noise levels will display more than the expected [1] amount of loss; on the other hand, however, we are unable to state what noise level over how long an exposure period will produce this excess, which persons will undergo a loss of how much, and, finally, how the loss will progress if the exposure conditions are maintained. These questions merely reflect the fact that we have qualitative but inadequate quantitative information about noise-induced hearing loss. The circumstance that industry now seeks answers to these questions indicates that we need the quantitative information.

In the collection of the necessary information, it seems to me that certain requirements must be met; these apply to two categories, the exposure variables and the subject variables.

Exposure variables

The mean level of the noise during the time of study must be known within reasonable limits of accuracy.

The frequency characteristics of the noise, i. e., its energy distribution with respect to frequency, should likewise be known.

The length of time during which any subject is exposed must be known within reasonable limits.

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1. The "expected" amount from the age group has been estimated from population studies, such as those reported by Beasley, W. C.: National Health Survey, Hearing Study Series, Bulletin 5, Table 3, United States Public Health Service, 1936, and Steinberg, J. C.; Montgomery, H. C., and Gardner, M. B.: Results of World's Fair Hearing Tests, J. Acoust. Soc. Am. **12**:291-301, 1940.

Subject variables

The subjects should be persons whose hearing acuity lies within the limits of normal hearing at the beginning of study.

The medical history and examination of these subjects should rule out incipient ear disease and/or other known predisposition to hearing impairment.

The subjects should preferably be chosen from the young-adult age level.

These subjects should be employed under conditions which involve frequent exposure to high-level noises.

The subjects should be selected from a trade or occupation in which the labor turnover is low.

It has been possible to approach these requirements in a study of shipyard employees. The sample chosen was selected from the apprentice groups at Mare Island Naval Shipyard, Vallejo, Calif., a field activity of the Bureau of Ships, United States Navy Department. The study was approved by the Office of the Surgeon General and has received the most wholehearted cooperation from all personnel concerned at the shipyard, including the Industrial Relations Department particularly, the Safety Office, the Apprentice Training School, the Industrial Laboratory, and the Medical Department.

TABLE 1.—*Noise Level Data* *

Area	Spectrum Distribution		
	Low	Flat	High
3	86.8	....	....
7	99.5	....	....
5	....	100.4	....
6	....	99.5	....
8	....	94.0	....
1	....	....	99.0
2	....	....	90.5
4	....	....	100.1

* Mean level in decibels.

STUDY

Noise Exposure.—A survey of the shipyard indicated eight areas in which prevailing noise levels either frequently rose above or consistently were above 90 db. A routine schedule of noise-level measurement was, therefore, set up, and data were systematically collected over a six-month period to date. In each of the eight areas multiple measuring points were chosen; in the largest area, for example, there were 18 such points. It was intended to accumulate a number of noise-level measurements sufficiently large to allow calculation of the probable mean level during any given interval.

The noise-level data consist of over-all level measurements and spectrum analyses taken, respectively, with a standard sound-level meter and a high-speed level recorder. A summary of these data is shown in Table 1. For convenience, the spectrum analysis has been divided into three categories: low—noises whose energy distribution is principally restricted to the lower end of the auditory range, flat—noises whose energy is approximately distributed equally throughout the auditory range, and high—noises whose principal components lie in the upper end of the auditory range.

Apprentices.—Men in the young-adult age group who are learning the several trades and specialties of shipyard service are apprentices. Their training requires them to spend considerable time in noisy areas, and this time can be accounted for. Each apprentice takes a four-year course, 25% of which is spent in the Apprentice Training School where regular classes are held. Because this four-year course is a requirement and also because each apprentice is being fitted for a

particular trade most applicable to the shipyard service, the turnover is very low, both during and after the years of apprenticeship. Under these conditions, one may hope for a sample the members of which will be available for study in a known environment for a long period of time.

The periodic classroom assignment (one week in each four-week interval) is advantageous, since it allows opportunity for the collection of resting audiograms uncontaminated by a superimposed temporary threshold shift.[2] The apprentices to be studied were, therefore, selected from the fourth and third classes, i. e., men in their first or second year of apprenticeship. These two classes included a total of 78 men.

Audiometric Conditions.—The library of the Apprentice School was found to be reasonably quiet, with an average noise level of 45 db. on the A network of the sound-level meter. Binaural

TABLE 2.—*Means of Thresholds at Each Frequency for Entire Group*

Frequency	250	1000	2000	3000	4000	6000	8000
Left ear	9.0	11.3	8.6	10.3	15.8	17.8	8.9
Right ear	8.7	12.6	7.7	7.0	12.6	16.1	7.2

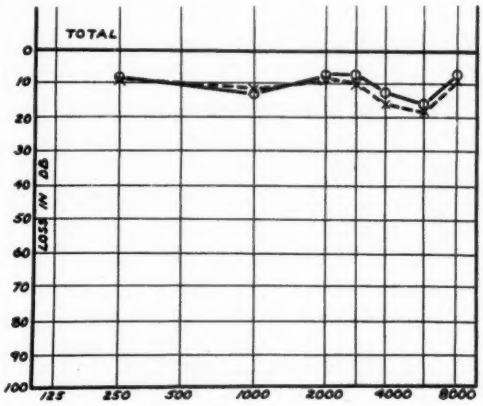

Fig. 1.—Mean audiogram of entire group.

receivers were used routinely, in an effort to minimize room noise still further. A certain amount of this noise was due to heating-system sounds. Each audiometer receiver was calibrated on an artificial ear before use, and output correction values at each frequency were obtained, in terms of the levels recommended by the National Bureau of Standards.

Audiometric Findings.—An audiogram was taken on each of the 78 men in the group. The time of measurement was set during a school week. A brief investigation of each man's history of noise exposure was made, with particular attention given to military or naval service. The thresholds at each frequency have been averaged for the 78 men and the means shown in Table 2.

The mean audiogram is shown in Chart 1.

It is evident from Chart 1 that the group contained a substantial proportion of men whose audiometric acuity was below normal. All audiograms, therefore, in which a loss was measured in excess of 15 db. at any frequency in either ear were culled out; there were 43 such audiograms rejected. The thresholds at each frequency have been averaged for the 43 men and the means shown in Table 3.

2. Wheeler, D. E.: Noise-Induced Hearing Loss, Arch. Otolaryng. **51**:344-355 (March) 1950.

TABLE 3.—*Means of Thresholds at Each Frequency for Group Rejected from Study*

Frequency	250	1000	2000	3000	4000	6000	8000
Left ear	10.3	12.3	11.3	15.2	25.1	28.9	19.4
Right ear	9.6	14.0	9.1	10.2	18.2	25.3	15.9

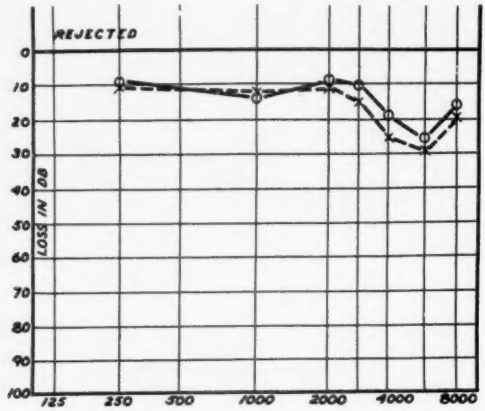

Fig. 2.—Mean audiogram of group rejected.

TABLE 4.—*Means of Thresholds at Each Frequency for Study Group*

Frequency	250	1000	2000	3000	4000	6000	8000
Left ear	7.3	9.6	5.2	4.0	3.6	3.2	—4.1
Right ear	7.6	10.7	5.8	3.0	5.0	4.1	—3.5

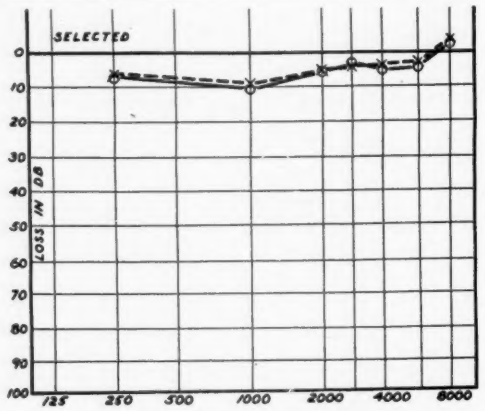

Fig. 3.—Mean audiogram of group selected.

The mean audiogram of the rejected group is shown plotted in Chart 2.

The remainder of the original group, 35 men, had audiograms considered normal. Medical examination of the ears, noses, and throats of these men disclosed no conditions predisposing them to ear diseases. Accordingly, this group was selected as the sample best fitting the requirements for future study. The thresholds for the selected group have been averaged and the means shown in Table 4.

The mean audiogram is shown plotted in Chart 3.

It is interesting to note certain properties common both to the group rejected and to the group selected. These are summarized in Table 5, prepared from information brought out in the individual histories.

TABLE 5.—*Properties Common to Group Rejected and Group Selected*

	Years	
	Rejected	Selected
Mean age	25.0	26.0
Age range	18-37 (79% < 30)	17-40 (71% < 30)
History of Noise Exposure		
Service	3.3	3.3
Other	2.7	2.8

TABLE 6.—*Means of Thresholds at Each Frequency on Reexamination of Study Group*

Frequency	250	1000	2000	3000	4000	6000	8000
Left ear	2.9	4.7	2.5	2.1	5.1	−0.9	−4.4
Right ear	5.6	5.8	2.1	0.1	9.1	1.3	−1.2

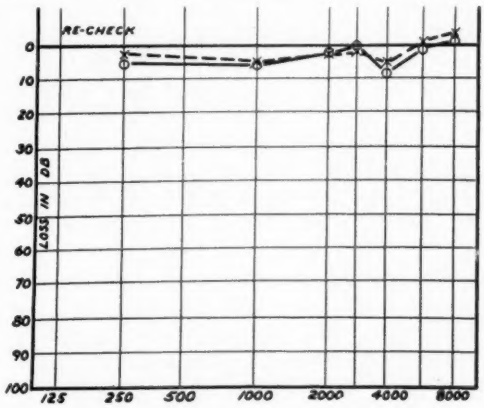

Fig. 4.—Mean audiogram of group selected; recheck.

Clearly, these groups are very similar in age and noise history.

Recheck.—The sample selected was reexamined after a six-month interval; at this time, 27 men were available.[3] The audiometric conditions were as before, with two exceptions: (1) absence of room noise due to heating system and (2) improved headband, permitting better fitting of audiometer receivers. The thresholds at each frequency have been averaged for the 27 men and the means shown in Table 6.

The mean audiogram is shown plotted in Chart 4.

In the recheck, the apparent slight improvement in group hearing acuity at all frequencies except 4000 and 8000 cycles must be interpreted with caution. It should be noted that the data reported here are the first of a necessarily long series; the trend, therefore, will depend on many additional measurements. The direction of the fluctuation shown at this time could be explained by several factors: (a) better-fitting receivers in recheck, (b) reduced room-noise level in recheck, and (c) fewer seasonal ailments (recheck conducted during summer).

3. It was to be expected that the number would vary somewhat during the course of the program, owing to leaves, absences, or temporary reasons.

COMMENTS

The possibility must be kept in mind that the group selected was not a random sample with respect to noise; the sample was constituted of persons whose ears have, apparently, withstood successfully considerable exposure to noise. Their ears, therefore, might be expected to show resistance to noise-induced loss in the future. Statistically, this sample is biased; it may not truly represent the industrial population. Since, however, this study is one of a number, it should be possible to evaluate the effect of potential bias as information is accumulated.

It would be prudent to point out that the plan described in this paper cannot reasonably be expected to realize entirely the objective listed in the introductory paragraphs. The paper is, rather, proposed as one in a series tending to establish a systematic and quantified body of knowledge about noise-induced hearing loss. Other studies are already in progress at other locations as a portion of the activities of the Committee on Conservation of Hearing (of the American Academy of Ophthalmology and Otolaryngology) through its Sub-Committee on Noise in Industry.

VOCAL-CORD PARALYSIS IN HEART DISEASE

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AND
MORRIS J. BROOKS, M.D.
BROOKLYN

THE ASSOCIATION of hoarseness and heart disease is a well-defined, although uncommon, clinical syndrome. Nevertheless, this combination has not received the attention it merits in the laryngological literature, either text or periodical. Two illustrative cases will, therefore, be included in this article, together with a brief résumé of the literature and a discussion of the pathogenesis.

The first description of this condition was given by Ortner in 1897.[1] Since then a number of articles on the subject have been published, but the first allusion to this association in a journal of laryngology was in the report of New and Childrey in 1932.[2]

The syndrome consists of hoarseness and enlargement of the heart. The hoarseness is the result of palsy of the left recurrent laryngeal nerve, and laryngoscopy may reveal any grade of defective movement of the left vocal cord, from mere sluggishness to complete paralysis. Involvement of the right side is rare, if seen at all.

The commonest form of heart disease involved is mitral stenosis. Other forms have been described,[3] but in all cases there was gross enlargement of the heart or of one of its chambers. The forms of heart disease which do not cause cardiac hypertrophy in some form do not produce left laryngeal nerve palsy. Often heart failure and dilatation or displacement of the pulmonary vessels are associated.

The presenting complaint is commonly hoarseness. In most cases the patient has already been receiving treatment for cardiac disease, but, occasionally, as in our second case, the existence of a heart disorder may have been previously overlooked or forgotten. In these cases, it is particularly easy not to recognize the syndrome and to attribute the symptoms to more sinister causes. The laryngologist may refer the patient to the internist with the suspected diagnosis of a mediastinal tumor. When the laryngologist learns the correct diagnosis, he may, with relief, transfer further treatment to the internist and, all too often, abandon his interest in the patient. As is illustrated in both cases presented here, the lesion is dynamic rather

Dr. Plotz is Associate Clinical Professor of Medicine, State University of New York, Medical Center at New York.

1. Ortner, N.: Recurrenslähmung bei Mitralstenose, Wien. klin. Wchnschr. **10**:753, 1897.

2. New, G. B., and Childrey, J. H.: Paralysis of the Vocal Cords, Arch. Otolaryng. **16**:143 (Aug.) 1932.

3. (*a*) Thompson, J. L., Jr., and Kistin, A. D.: Hoarseness in Heart Disease, Ann. Int. Med. **29**:259, 1948. (*b*) Diefenbach, W. C. L.: Left Vocal-Cord Paralysis Associated with Hypertensive Heart Disease, New England J. Med. **240**:419, 1949.

than static with regard to both the heart and the laryngeal conditions. Neither the laryngologist nor the internist should relinquish his concern for the patient.

Treatment is only that of the underlying heart disorder. There may be complete, little, or no return of function after therapy. The cases which are the result of cardiac or great-vessel dilatation seem to respond better than those in which there is gross enlargement of one chamber, a condition that is usually irreversible. The commonest example of the latter is the enlargement of the left auricle in mitral stenosis.

The cause of the injury to the left recurrent laryngeal nerve has not been definitely established, but numerous theories have been advanced and were recently summarized by Diefenbach.[3b] In 1911 Fetterolf and Norris [4] studied frozen specimens and concluded that dilatation of the left auricle caused the left pulmonary vein to impinge on the pulmonary artery, which in turn was forced against the aorta, the nerve being compressed between the left pulmonary artery and the aorta or ligamentum arteriosum. Kraus [5] blamed dilatation of the right ventricle, which displaced the heart to the right, dragging on the aortic ligament, thus stretching the nerve. Guttman and Neuhof [6] thought that the nerve might be incorporated between bands of pericardial and mediastinal adhesions. This may have been true of their case, but the explanation is not acceptable for other patients. King, Hitzig, and Fishberg [7] reported the syndrome in arteriosclerotic heart disease with left ventricular failure. They emphasized, for the first time, the possible role of dynamic dilatation of the pulmonary vessels in causing compression of the nerve. Dolowitz and Lewis [8] felt that

> lymph nodes in the triangle formed by the pulmonary artery, aortic arch and ligamentum arteriosum may effectively compress the left recurrent laryngeal nerve when accompanied by cardiac hypertrophy, pulmonary artery engorgement, or both.

The usual course of events in the larynx is involvement of the abductor muscles, then of the tensor and adductor muscles, and, finally, total hemilaryngoplegia. The course may be arrested at any point short of complete paralysis.[9]

During the first stage (abductor impairment alone), there may be no hoarseness and the lesion is overlooked, since there are no presenting symptoms. Later, the unopposed action of the adductor muscles and the onset of paralytic contracture maintain the cord in or near the midline even on deep inspiration, while the tensor muscle keeps the cord taut. With the other cord working freely, the voice may, therefore, be unimpaired; at this stage, the diagnosis is frequently missed because usually no dyspnea is noted, except possibly on exertion. Our Patient 1 had no hoarseness and even a clear singing voice at the time of her most recent examination when the conditions just described were discovered.

4. Fetterolf, G., and Norris, G.: Anatomical Explanation of Paralysis of Left Recurrent Laryngeal Nerve Found in Certain Cases of Mitral Stenosis, Am. J. M. Sc. **141**:625, 1911.

5. Kraus, cited by Fetterolf and Norris.[4]

6. Guttman, J., and Neuhof, S.: Radial Pulse Difference and Left Recurrent Nerve Paralysis Due to Mitral Stenosis, J. A. M. A. **66**:335 (Jan. 29) 1916.

7. King, F. H.; Hitzig, W. M., and Fishberg, A. M.: Recurrent Laryngeal Paralysis in Left Ventricular Failure, Am. J. M. Sc. **188**:691, 1934.

8. Dolowitz, D. A., and Lewis, C. S.: Left Vocal Cord Paralysis Associated with Cardiac Disease, Am. J. Med. **4**:856, 1948.

9. Thomson, St. C.: Diseases of the Nose and Throat, ed. 3, New York, D. Appleton & Company, 1927, p. 597.

REPORT OF CASES

CASE 1.—G. A., a white housewife 49 yr. of age, reported for cardiac survey with the presenting complaint of swelling of the ankles for one month. There was no history of rheumatic fever, but cardiac murmurs were heard on routine examination 18 years before. No symptoms were noted until 12 years later, when there was moderate exertional dyspnea and examination revealed auricular fibrillation and liver enlargement. The patient had taken digitalis intermittently since then. Edema of the legs appeared for the first time just before she was observed by us.

The cardiac findings were similar to those noted in Case 2, i. e., signs of mitral stenosis, except for the added element of mitral regurgitation and enlargement of the left ventricle. A widely transmitted systolic apical murmur and a much more limited diastolic murmur were present. The rhythm was that of auricular fibrillation. Roentgen examination showed enlargement of the left auricle and ventricle, with encroachment on the esophagus and lifting of the

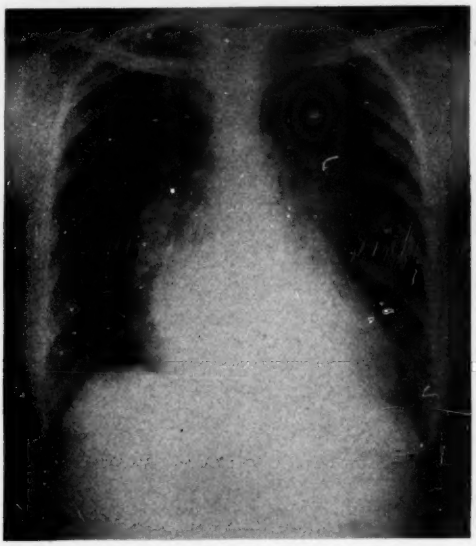

Fig. 1 (Case 1).—Teleroentgenogram. The left ventricle is enlarged and the left pulmonary conus pushed up.

left main bronchus. There were a few rales at the bases of the lungs, edema of the ankles, and hepatomegaly.

It was noted during the heart examination that the patient was hoarse. She reported that the onset of hoarseness coincided almost exactly with the appearance of ankle edema and the general worsening of her cardiac status. It is possible that a rather abrupt increase in cardiac dilatation occurred at that time.

Laryngological consultation disclosed the voice to range from slightly strident to almost normal. It became strident rapidly when in use. On quiet respiration, the left cord was in the cadaveric position, with slight concavity. On forced breathing minimal abduction was present. On phonation there was only slight adduction. The right cord compensated by crossing the midline, the right arytenoid cartilage rotating anteriorly in front of the left.

The course was more favorable than that in Case 2. With the renewed use of digitalis, a salt-poor regimen, and dehydration therapy, her condition improved rapidly and the cardiac

symptoms disappeared. The voice improved promptly, and the patient is now able to sing well. There is no longer any concavity of the left cord, which appears taut, and there is practical approximation of the cords. The left cord is, however, fixed in position.

CASE 2.—A. S., a white male accountant aged 38, reported to the laryngologist, complaining of hoarseness. Results of examination of the head were irrelevant except for the laryngeal findings. The voice was hoarse with a low pitch and strident with audible hissing as the air escaped.

Laryngoscopy showed that during quiet breathing the left cord was between the normal positions for forced and quiet breathing. During forced breathing, there was only minimal further abduction of the left cord. On phonation (high-pitched "E"), the left cord moved halfway to the midline, with the right cord overcompensating to close the gap almost completely.

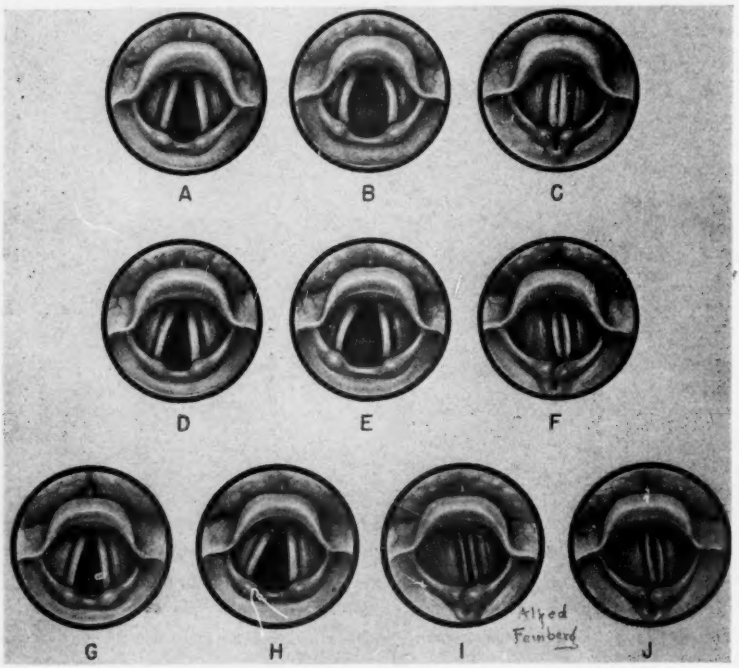

Fig. 2.—Normal larynx: *A*, quiet breathing; *B*, forced breathing; *C*, phonation: "eee." Case 1: *D*, quiet breathing; *E*, forced breathing; *F*, phonation: "eee." Case 2: *G*, quiet breathing; *H*, forced breathing; *I*, phonation: "aaa"; *J*, phonation: "eee." Phonation of "aaa" (*I*) is represented as extreme compensatory effort of right cord by its tautness and torsion, so that the anterior third is curled on itself.

The right arytenoid cartilage passed in front of the left, giving the appearance of good approximation in this area, with a rectangular chink anteriorly. At the end of phonation the right cord abducted with alacrity; the left, slowly. There was slight concavity of the left cord, with no apparent loss in tension. The diagnosis of secondary paresis of the left recurrent laryngeal nerve was made, and the patient was advised to have a diagnostic survey. He was admitted to the Long Island College Hospital.

Physical examination showed nothing of importance except in the larynx and heart. There was typical mitral stenosis. A short presystolic crescendo murmur was heard over a small area just within the apex, which was not displaced. The first sound at this point was loud. The

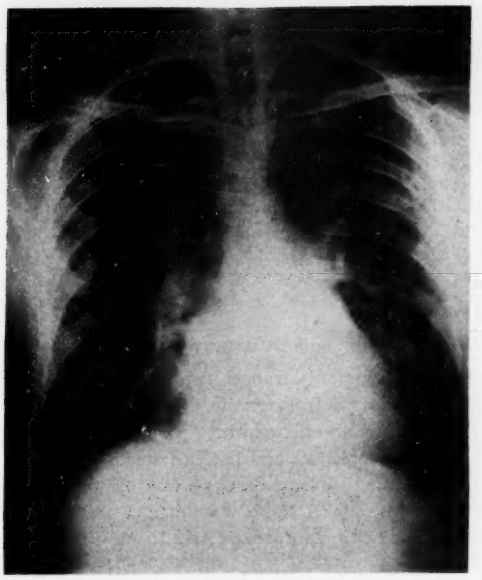

Fig. 3 (Case 2).—Teleroentgenogram showing mitral stenosis. The left auricle is enlarged and the left pulmonary artery pushed up.

Fig. 4 (Case 2).—Esophagram (barium-sulfate suspension) taken in the right oblique position. The left auricle is much enlarged, displacing barium-filled esophagus.

second pulmonary sound was accentuated. Fluoroscopy and roentgenography failed to reveal any mediastinal neoplasm. The left auricle was enlarged, encroaching on the barium-sulfate-filled esophagus. The left pulmonary artery was displaced. The left main bronchus was elevated and compressed.

It is noteworthy that the patient had no symptoms or other signs referable to heart disease. There was no history of rheumatic fever, dyspnea, or other disability, and he was shocked to learn that he had heart disease. Within a year, however, the heart rhythm changed, after a cold, to that of auricular fibrillation, and moderate heart failure supervened.

At the present writing (about two years after he was first seen), the patient continues his work but must take digitalis. Generally speaking, there has been little change in his voice, with a slight deterioration of the paresis. It should be noted that there have been several partial remissions lasting a week or two at a time, but the course has been unsatisfactory.

SUMMARY

Paralysis of the left recurrent laryngeal nerve may occur in combination with heart disease, most commonly mitral stenosis. The cause of the paralysis is probably compression of the nerve by an enlarged or displaced left pulmonary artery.

Two cases of severe hoarseness and left hemilaryngoplegia in association with mitral stenosis are presented. In one case, no improvement was noted on therapy for cardiac disease; in the other, there was distinct amelioration of both cardiac and laryngeal symptoms with appropriate treatment directed to the disorder of the heart.

PREVENTION OF OTITIS IN THE SWIMMER

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IT HAS been my endeavor for some time to find an efficient method for prevention of the otitis resulting from swimming or bathing, with particular emphasis on those problems caused by water entering the external ear canal alone, or entering the external ear canal and the middle ear via a perforation in the eardrum membrane.

Interest in this study was stimulated primarily by two types of cases: first, that of the patient with a perforation of the eardrum membrane, which the otologist is attempting to close by repeated treatments, who, in the midst of treatment, goes swimming or gets water into the middle ear during a shower bath, with ensuing infection and delay in the treatment; second, that of the patient with external otitis whose complaint was apparently initiated or is aggravated by water entering the ear canal.

That water may also enter the middle ear via the Eustachian tube is not to be denied.[1] Nielsen [2] stated, in writing on swimmer's otitis, that in 90% of the patients he examined the otitis was caused by pneumococci which, he felt, were endogenous, arising from nasopharyngeal bacteria, and that impure bathing water played no etiologic role. Nielsen called attention to the fact that water forced through the nose into the nasopharynx washes away the protective layer of mucus over the cilia of the nasal mucosa and thus retards or completely abolishes ciliary activity. Damage to nasal mucosa was demonstrated microscopically by Mezz in 1940.[3] Nielsen's technique in his 1945 series was that of aspirating the tympanic secretion after myringotomy, thus avoiding the contaminating flora of the external auditory canal.

As bacteriological examination of the water of Copenhagen swimming pools showed no pneumococci, Nielsen discarded the thesis that impure water is an etiologic factor. He believed that the infection occurred by way of the Eustachian tube, and he explained the fact that a pure pneumococcic infection and not a mixed one

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A résumé of this material was presented at a meeting of the Philadelphia Laryngological Society, March 6, 1951.

Thesis approved by the faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Master of Medical Science (M.Sc. [Med.]) for graduate work in otolaryngology.

1. Coates, G. M.: Lecture on Otitis Media in Graduate School of Medicine, University of Pennsylvania, 1947.

2. Nielsen, J. C.: Swimmers' Otitis: Etiology, Symptomatology, and Pathogenesis, Acta oto-laryng. **33**:31, 1945.

3. Mezz, D.: Otorhinologic Sequelae of Swimming, Laryngoscope **50**:479, 1940.

was present by the hypothesis that the pneumococci in the nasopharynx are easily affected by general and local reduction in body temperature.

Gleason [4] stated that when the tympanic membrane is intact fluid enters the narrow Eustachian tube beyond the isthmus only with great difficulty, having to compress before it the air contained in the middle ear. As soon as the pressure is relaxed, the spring or rebound of the compressed air generally throws into the pharynx the fluid contained in the tube. However, during the act of swallowing, fluid may be made to penetrate into the cavity of the tympanum through the Eustachian tube even when the tympanic membrane is intact, the muscular action in opening and shutting the tube during swallowing doubtless playing an important role in such circumstances.

In this manner, Gleason wrote, sea water or fresh water introduced into the pharynx while bathing sometimes reaches the tympanum and almost invariably produces acute otitis media. He also stated that he had observed the same occurrence during the use of the Birmingham douche or, even, from the sniffing of isotonic sodium chloride solution into the nose from the hollow of the hand.

To prevent this type of otitis media, one should try to keep the water from getting back as far as the Eustachian tubes. The swimming instructor can help a great deal with timely advice to his students. Also important is the use of a nose clip.

Swimming Coach Steven Forsyth [5] declared, "Strange as it may seem, maintaining normal breathing while swimming is more difficult than learning the mechanics of the stroke." Today, most instructors teach primarily the "American six-beat crawl stroke," which is a head-under-water stroke. The air is inhaled through the mouth and exhaled through the nose into the water. "To time a complete respiration in any style of swimming without holding the breath during any part of the stroke is a real art," wrote Coach Forsyth. "Any jerky motion is associated with faulty breathing." He continued:

> Human response to practically every emotion first registers in the respiratory rate; therefore, all of the common emotions such as fear, anxiety, worry, affect the breathing. . . . Every lesson starts out with bubble blowing—I have my veteran swimmers do it, too. It is difficult at first to exhale a complete breath in the water but it comes easily with practice.

Grace B. Davis,[6] Assistant Professor of Physical and Health Education at the University of Cincinnati, was most emphatic in her teaching of the importance of proper breathing in swimming and diving in order to prevent water entering the sinuses and the postnasal space. With forced exhalations of air through the nose, properly timed, water cannot reach the sinus openings or postnasal space, according to Prof. Davis' teaching. She even had her pupils exhale in diving to prevent water from reaching the interior of the nose.

For the untrained swimmer, the nasal clip is probably the best solution for this problem. It closes off the nose entirely and is not uncomfortable to wear. It also protects the sinuses.

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4. Gleason, E. B.: A Manual of Diseases of the Nose, Throat and Ear, ed. 7, Philadelphia, W. B. Saunders Company, 1933, p. 359.

5. Forsyth, S.: Quick Way to Better Swimming, Garden City, N. Y., Sun Dial Press, Inc., 1939.

6. Davis, G. B.: Sinusitis and Ear Infections as Related to Swimming and Diving, J. Health & Phys. Educ. **9**:308, 1938.

But these two means of prevention rarely come within the province of the otologist. The part that the physician usually plays is in the prevention of the type of trouble that results from water entering the external ear canal. Is this trouble usually due to the contamination in the water or to factors in the human body itself?

DISEASE TRANSMISSION BY BATHING WATERS

Let us first examine the question of the extent and prevalence of disease transmission by bathing waters. In discussing this, consideration must be given to those diseases which might be spread from one bather to another and to those diseases which might be caused by the pollution of bathing waters from public sewerage systems and other extraneous sources.

The usual types of diseases considered in connection with bathing waters are eye, ear, nose, and throat infections, skin infections, such as ringworm and scabies, venereal infections, and gastrointestinal diseases.[7] Despite all the questionnaires and data collected by the American Public Health Association, it must be admitted that there is a great dearth of epidemiological information about just what sickness is caused by bathing in polluted water. Surveys have revealed surprisingly little reliable data to indict bathing places in the spread of diseases.

Conditions pertaining to bathing waters are decidedly different from those pertaining to water supplies. Many persons swallow little or no water during bathing—particularly is this true of salt water—and there is a fairly rapid change of water at the majority of bathing places. These conditions tend to reduce the possibilities of disease spread from bathing places.[8]

Considerable interest has been evidenced by many public-health workers in the possibilities of spread of skin infections from bathing. Occasionally, cases of skin eruptions on the bodies of bathers have been reported. In Wisconsin and Michigan extended studies have been made of the occurrence of "schistosome dermatitis," or "swimmer's itch," which has affected bathers in the waters of lakes, rivers, and ponds, or beach pools which support snails. The causative agents are the cercariae, the free-swimming larval stage of certain parasitic worms of the family Schistosomidae, the snails serving as an intermediate host. Authorities in both Wisconsin and Michigan have carried out treatments of infected bathing places. The studies of control of aquatic growths by the Wisconsin State Board of Health and other agencies in that state are particularly worthy of mention. A committee on water pollution published in July, 1939, a progress report on the chemical treatment of lakes and streams, with special reference to the origin and control of swimmer's itch. Warrick, cited in the handbook of the American Public Health Association,[8] reported that satisfactory results were obtained on treatment of aquatic growths with copper sulfate for algae and cercariae control and with sodium arsenite for rooted weed control. Copper carbonate also appeared to be quite effective for suppressing the snail population. The fish were not harmed by the amount of chemicals used.

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7. Hitschler, W. J.: The Relationship of Swimming and Diving to Sinusitis and Hearing Loss, Laryngoscope **59**:799, 1949.

8. American Public Health Association: Recommended Practice for Design, Equipment, and Operation of Swimming Pools and Other Public Bathing Places, New York, The Association, 1942.

The American Public Health Association [8] stated that eye, ear, nose, and throat infections have been reported.

Some of these might be due to lowering of resistance from chills; water may wash away protective mucous discharges containing anti-bodies, leaving the affected parts susceptible; harmful organisms already present in the body may be washed further into the ear and nasal passages during bathing; or excessive amounts of chemicals used for water purifying may cause inflammation of mucous membranes and irritation of the skin. These are all possibilities aside from any harmful infections in the water.

In the light of the recently reported recovery of the virus of poliomyelitis from the intestinal tract and from sewage, a question has been raised whether this disease might result from bathing in sewage-polluted water. However, there has been no actual indictment of the bathing place as a causative agent in the spread of poliomyelitis, even though the subject may deserve further research and study. To date, there has been no published information to show what effect, if any, sewage-treatment-plant processes have on the destruction or disappearance of the virus.

The American Public Health Association is unconvinced that bathing places are a major public health problem, even though bathing-place sanitation, because of the health considerations involved, should be under careful surveillance of the public-health authorities, and proper sanitary control of bathing places should be exercised.

Major health problem or not, otologists do know that many ear problems arise in connection with bathing.

TYPES OF BATHING PLACES

What then are the different types of bathing places? For the purposes of this report, they may be said to include all bodies of water sufficiently deep for complete immersion of the body. These places, together with their shores, buildings, equipment, and appurtenances, are usually visited collectively by numbers of persons for swimming or recreative bathing.

Bathing places may be divided into three classes:

1. Natural outdoor ponds, rivers, tidal waters, etc.
2. Outdoor pools which are partly artificial and partly natural in character.
3. Pools, outdoor or indoor, which are entirely of artificial construction.

Natural ponds and rivers are necessarily dependent upon natural flow or upon temperature, wind and wave action for circulation of the water. Artificial and partly artificial pools may be divided into four classes according to the method by which water cleanliness is obtained:

a. Fill and draw pools where cleanliness of water is maintained by complete removal and replacement of the water at periodic intervals.

b. Large semiartificial pools in which water cleanliness is maintained by natural flow or circulation in a manner corresponding to that of a natural pond or lake.

c. Flowing-through pools where cleanliness is maintained by circulation of water through the pool from some natural or developed source, but where the outflowing water is wasted.

d. Recirculation pools in which circulation of the water is maintained through the pool by pumps, the water drawn from the pool being clarified by filtration before being returned.[8]

CHARACTER AND TREATMENT OF WATER

Let us digress momentarily to consider something of the character and treatment of water.

Water is a liquid having a blue color, and it dissolves most substances, even rocks and metals, to a greater or less degree.

Water, from a sanitation engineer's point of view, is divided into these classes [9]:

1. *Meteoric Water.*—All water derived directly from the atmosphere is called meteoric water, whether in the form of dew, rain, snow, sleet, or hail. Water evaporated from fresh-water lakes and condensed on high mountains is exceptionally pure, but rain and snow collected in towns are more or less polluted with the washings of air and roofs, especially during the beginning of storms. Rain water should be collected from clean surfaces, and the first runnings from the collecting surface should be rejected. Rain water usually has a mawkish taste which may be overcome by filtration and aeration. It should be filtered before being used for household purposes.

2. *Ground Water.*—The meteoric water which sinks into the ground and flows seaward along more or less impenetrable stratums is called ground water—subdivided for convenience into spring, shallow well, deep well, and artesian water.

Appearance: Meteoric water falling upon the earth and passing through thick enough layers of proper soil loses its suspended matter and becomes clear. An exception is water carrying fine particles of clay with it through the relatively large pores of water-bearing soil or the fissures and seams in rock. Waters which become turbid after rains are usually polluted from surface sources. Some waters which are clear when drawn become turbid on standing because of the precipitation of iron, manganese, calcium and manganese carbonate, etc., which generally follows the escape of the gases that hold these substances in solution. Ground waters are usually colorless, but not always so.

Efficiency of Natural Purification: The distance which a water must pass through the soil before becoming safe is important and depends upon local conditions, such as rate of flow, character of soil, and degree of pollution. Again the work imposed upon the soil may exceed its capacity; aerobic bacteria may be deprived of oxygen and give way to anaerobic bacteria, a condition not so favorable to bacteriological purification of ground water. A well-aerated soil is much more efficient than a soil which has been robbed of its oxygen.[9]

The numbers of bacteria in soil and in ground water decrease rapidly with depth. Kabrhel [10] found several million organisms per cubic centimeter in surface samples of woodland soil, a few thousand organisms per cubic centimeter 0.5 m. below the surface, and usually only hundreds per cubic centimeter in samples collected from depths of greater than 1 m. Many organisms in ground water grow slowly and, therefore, do not appear after short periods of incubation, thus giving rise to the erroneous belief that ground waters from considerable depths are invariably bacteria-free. Prescott [11] examined 147 shallow farmyard wells and found that 124 contained no colon bacilli and were, therefore, probably free from fecal pollution. The samples averaged 190 bacteria per cubic centimeter, while the 23 samples which gave positive reactions for E. coli averaged 570 bacteria per cubic centimeter.

9. Flinn, A. D.; Weston, R. S., and Bogert, C. L.: Waterworks Handbook of Design, Construction and Operation, New York, McGraw-Hill Book Company, 1916.

10. Kabrhel, G.: Bacteria in Ground Water, Arch. Hyg. **58**:345, 1906.

11. Prescott, S. C.: Bacteria in Well Water, in Prescott, S. C., and Winslow, C. E. A.: Elements of Water Bacteriology, New York, John Wiley & Sons, Inc., 1913, p. 27.

Kellerman and Whittaker [12] reported similar results for shallow wells used as farm water supplies.

Deep well waters contain few bacteria. Houston [13] reported the number of bacteria in the waters from a deep well of high quality at Tunbridge Wells, England, to vary from one to 36 per cubic centimeter. Prescott [11] found in the water of 15 driven wells in the vicinity of Boston an average of 18 colonies of bacteria per cubic centimeter at the end of 48-hr. incubation.

Colon bacilli, which are the normal inhabitants of the human intestine and of the intestine of various animals, are found in polluted ground waters. Colon bacilli also exist outside the bodies of animals, and organisms giving the reactions of E. coli may live in a semiparasitic fashion on plants as well as animals.[14] The term colon bacilli covers a number of forms or types; it really represents a whole class of bacteria. Therefore, it is the number of organisms found in ground water, as in surface water, rather than their mere presence, that is of sanitary importance. E. coli should be used most cautiously as a basis for judging the quality of water, largely because the "presumptive test" for these bacteria tends more and more to include organisms of the colon group as well as the typical bacillus of the human intestine.[9] E. coli may occasionally be found in ground waters of good quality, provided large enough volumes are tested. The bacilli abound at the ground surface, and a few may penetrate to great depths. That their mere presence proves the presence of infinitely rarer disease germs is inconceivable.[9] The permissible number of E. coli in a ground water is difficult to determine. E. coli should not be present in 1-cc. samples or in more than 20% of 10-cc. samples. E. coli will rarely be found in the best ground waters, even in 100-cc. samples, or in more than 10% of the 10-cc. samples from most undeniably safe sources. However, certain exceptional waters from the vicinity of peaty deposits, such as exist on Cape Cod and Long Island, contain bacteria showing most of the E. coli reactions. Such waters should not be judged by the reaction to the presumptive test for E. coli alone.[9]

3. *Surface Water.*—The most generally available sources for surface water are streams, rivers, ponds, and lakes. It is especially liable to pollution and, for this reason, is rarely suitable for domestic use without purification by storage, treatment with chemicals, filtration, or disinfection.

Organic material in surface water includes many kinds of suspended matter, both living and dead. This is sometimes called plankton, although the term is usually restricted to living organisms. Suspended organic matter includes that organized as microscopic plants and animals, the former including the bacteria, which are true plants, although some of them possess the power of locomotion. It also includes wastes of plant and animal life and organic matter discharged with sewage from factories and towns. Growth of organisms in surface water is dependent upon the presence of food and light. Nitrates and carbon dioxide form the ideal plant food and give rise to abundant growths of algae. Bacteria which feed upon or peptonize the suspended matter and bacteria which mineralize the dissolved organic matter, with the algae, furnish food for growth of Protozoa; these, in turn,

12. Kellerman, K. F., and Whittaker, H. A.: Bulletin 154, United States Department of Agriculture, Bureau of Plant Industry, 1909.

13. Houston, A. C.: Report on the Chemical and Bacteriological Examination of Tunbridge Wells Deep Well Water, Rep. Med. Off. Local Gov. Bd. 1902-1903, London, 1904, p. 581.

14. Prescott, S. C.: B. Coli in Ground Water, Science **15**:363, 1915.

form part of the food for growth of Hydrae and Rotifera, all of which are consumed by fish. Oysters and clams also feed upon diatoms and other microscopic organic matter, living and dead, and the abundance of the higher plant life supported indirectly by the organic matter contained in water in which it grows is well known. Often the only apparent result of increased organic pollution of a body of water is in a corresponding increase in fish, oysters, clams, etc., as for example, in the Illinois River [15] and the Genesee River.[16]

Fecal waste from the higher animal forms decomposes into substances which, in the presence of oxygen, pass through the nitrogen cycle. Some organic matter present in a water is removed in the form of the adult insects whose larvae live in the water: for example, Chironomas. Solid masses of organic sludge in the bottoms of streams become gradually liquefied by anaerobic bacteria. These sludge masses also serve as food for a variety of worms and larvae, but only in the presence of oxygen. Liquefied sludge forms part of the food of bacteria and other organisms. There exists, therefore, in many streams containing undecomposed organic matter, a continuous cycle of changes, resulting ultimately in biological equilibrium and, if no new food material is forthcoming, in rapid decadence of organic life and mineralization of the dead organic matter, i. e., to nitrates, nitrites, carbon dioxide, water, etc. In other words, growth of organisms, including bacteria, plays the most important role in self-purification of streams.[12]

The surface waters of most rivers in inhabited regions contain several hundreds or thousands of bacteria per cubic centimeter, dependent upon the degree of pollution. Furthermore, these numbers fluctuate rapidly, especially during storms and floods.[17]

In warm weather and during periods of low flow, putrefaction is likely to occur in river water, greatly changing its bacterial count. This condition is accompanied with loss of oxygen, sometimes its entire disappearance and the destruction of fish life, which may indirectly cause an increase in the bacteria. Temperature itself has slight influence upon the numbers of bacteria, although growths of antagonistic microscopic organisms are most frequent during the warmer months.[9]

Lake waters contain fewer bacteria than river waters, although lakes are polluted with sewage at the shores and at the mouths of rivers. Checks on the water from Lake Superior near Duluth, Minn., by Weston showed no bacteria at the depth of 100 ft. (30.5 m.), although the surface water, polluted by sewage from Duluth, contained several hundred per cubic centimeter. Dr. E. Channing Stowell, as reported by Flinn and others,[9] found the water of Dublin pond (New Hampshire) practically sterile at the depth of 40 ft. (12.2 m.), although colon bacilli were occasionally discovered along the shores. Lake Ontario near Toronto, Canada, is an example of polluted lake water.[18]

The chief sources of contamination are sewage of cities and towns and wilful or careless discharge of dejecta by human beings. It is interesting and informative to note that intestinal bacteria, including E. coli and the typhoid bacilli, tend to die out

15. Farber and Richardson: Organic Matter in Surface Water, in Flinn and others,[9] p. 658.

16. Whipple, M. C., and Bunker, J. W. M.: Bacteria in Surface Water, United States Public Health Service Reprint 710.

17. Whipple, G. C.: Microscopy of Drinking Water, New York, John Wiley & Sons, Inc., 1914.

18. Longley, T.: Trans-Canadian Society of Civil Engineers, Nov. 6, 1913.

in natural waters. According to various authorities,[19] more than 95% of typhoid bacilli discharged into surface water die during the weeks following their entrance, and after one month they have practically ceased to exist. The surviving minority may persist as long as two months. Experiments have shown that typhoid bacilli perish more rapidly in polluted surface waters than in unpolluted ground waters. Jordan, Russell, and Zeit [19b] attribute this destruction to the antagonism of normal water bacteria. Protozoa also consume bacteria.

From a sanitary standpoint, the discharge of many million bacteria by one infected patient into water about to be distributed is far more serious than the discharge of bacteria with the sewage of a city, if the contaminated water is stored for a month or two before use. Storage, in other words, tends to cut down the bacterial count if the water is kept from contamination. Long storage coupled with careful inspection can produce fairly safe water even from polluted sources.[9] It must be said, however, that the quality of water is greatly affected by storage, for both good and bad. Storage affords opportunity for coagulation, precipitation, bleaching of

TABLE 1.—*Principal Chemicals Used for Water Purification*

Trade Name	Strength	Formula of Active Ingredients
Basic sulfate of alumina (aluminum sulfate)....	16-23% Al_2O_3	$Al_2(SO_4)_3 + H_2O$ * + Al_2O_3
Ferrous sulfate	95-100% $FeSO_4 \cdot 7\ H_2O$	$FeSO_4 \cdot 7\ H_2O$
Copper sulfate	99% $CuSO_4 + 5\ H_2O$	$CuSO_4$
Quicklime (calcium oxide).....	75-99%	CaO
Hydrated lime (calcium hydroxide).....	80-99% $Ca(OH)_2$	$Ca(OH)_2$
Soda ash (sodium carbonate).....	58% Na_2O	Na_2CO_3
Caustic soda (sodium hydroxide).....	70-76% Na_2O	NaOH
Barium carbonate	98% $BaCO_3$	$BaCO_3$
Bleaching powder	30-38% Cl	$CaCl_2 \cdot Ca(OH)_2 \cdot H_2O$ + $Ca(OCl_2) \cdot 2Ca(OH)_2$
Liquid chlorine	100% Cl	Cl_2

* Uncertain number of water molecules; the substance is a mixture.

color, death of exotic disease germs, and subsidence of silt and clay. Sometimes, however, stored waters develop growths and microscopic organisms, especially if the sources contain an abundant supply of food material or the water is stored in a new reservoir from which the vegetation and other organic matter have not been adequately removed.

Storage reduces the total numbers of bacteria and also devitalizes disease germs.

Aeration.—Aeration consists in bringing water into intimate contact with air in order to introduce oxygen for the oxidation of iron or organic matter and for washing out gases and volatile odors.

Coagulation and Other Treatments.—Water containing less than 20 ppm of color can be purified by filtration through sand without coagulation. Filtration alone cannot completely remove colloidal clay, organic matter, and dissolved vegetable color. These are in colloidal solution and must be coagulated before filtration.

Basic sulfate of alumina is the most commonly used coagulant (Table 1).

19. (*a*) Gärtner, A.: Origin and Longevity of Polluting Bacteria, Klin. Jahrb. **9**:335, 1902. (*b*) Jordan, E. O.; Russell, H. A., and Zeit, F. R.: The Longevity of Typhoid Bacillus in Water, J. Infect. Dis. **1**:641, 1904. (*c*) Houston, A. C.: Studies in Water Supply, London, The Macmillan Company, 1913, p. 83 ff.

Ferrous sulfate in conjunction with lime is a good coagulant for turbid alkaline waters.

Copper sulfate is used as an algacide. Ten parts per million of copper sulfate (blue vitriol) will destroy colon bacillus.

Lime is used for water softening.

Soda ash (anhydrous sodium carbonate) is used to furnish alkali to waters which are deficient therein.

Barium carbonate is sometimes used to remove calcium sulfate from water.

Bleaching powder is an active sterilizing and oxidizing agent.

The disinfection of water is practiced to destroy disease germs. Total destruction of the organisms by sterilization is not usually considered necessary.[9]

Disinfection may be accomplished by heat, light, application of chemicals, including ozone, and ultraviolet rays. Heat is an efficient disinfecting agent, but it is too costly when large volumes of water have to be treated.

TABLE 2.—*Comparison of Antiseptics*: Tissue Toxicity and Bactericidal Effect*

Compound	Staph. Pyogenes var. Aureus	
	Toxicity Index†	Phenol Coefficient
Halogens		
Iodine	0.2	82.0
Chloroazodin	0.3	236.3
Phenols		
1, 3, dihydroxy-4-hexylbenzene	0.9	14.7
Mercurials		
Phenol		
4-nitro-5-hydroxymercuri-*o*-cresol anhydride	1.5	12.5
Sodium (ethylmercuri) thiosalicylate	169+	— 0.23
Silver compounds		
Silver nitrate	1.8	0.7

* From Salle, A. J.; McOmie, W. A.; Shechmeister, I. L., and Foord, D. C.: J. Bact. 37:639, 1937.
† Ratio of highest dilution preventing growth of embryonic tissue to highest dilution killing test organism (10-min. periods).

Oxidizing Agents.—Chlorine, bleaching powder, and sodium hypochlorite are the most efficient and economical chemical disinfectants. Their efficiency is roughly proportional to their chlorine content.

Light.—Sunlight exerts a powerful bactericidal action on the surface waters of reservoirs. This action, even in clear waters, is of no importance at depths of more than a few feet, unless the water is in active circulation and the action of light is exerted on successive surface layers. Light has practically no effect in turbid or highly colored waters.

Bacteriological examination for certain unicellular forms is important. Certain of these bacteria are normal inhabitants of all surface waters and some ground waters. Some bacteria are entirely harmless, some very harmful, and some indicative of possible presence of harmful forms. Each species has both its natural and its accidental habitat. Any examination of water does not reveal all the bacteria present; methods for this have not been discovered, but the number growing under standard conditions is an index of the amount of pollution or contamination and sometimes of the quantity of food material available for their growth. Fluctuations in the height of streams and general water conditions have a marked effect upon

the numbers of bacteria in surface waters. Of far greater importance, however, than mere numbers is the detection of certain organisms known to accompany fecal pollution.

Determination of the bacterial flora in water is not usually worth the labor involved, and, in most cases, gives no better idea of its sanitary condition than do the results of tests for E. coli.[20]

Fungi and Molds.—Fungi are flowerless plants without chlorophyll or starch. They are either saprophytic or parasitic. Fungi consist of a vegetable portion called mycelium, which forms the groundwork of common mold or mildew, and the fruit, which is borne at the ends of the branching filaments, composed of spore cells, sometimes highly colored. Well-known molds of the filamentous variety are Mucor, Penicillium, and Aspergillus. On investigations of water supplies the most important fungi to the engineer are Crenothrix and other so-called iron bacteria, which grow abundantly in waters containing iron and manganese. These organisms sometimes precipitate iron to such an extent that it may completely clog services and small mains.

An analytical examination of water consists of a series of tests and experiments, usually from 10 to 25, to assist in ascertaining the past history and the present condition of the water.

The following synopsis explains the significance of the various tests:

Color tests measure the colored substances in solution, such as vegetable matter dissolved from roots, leaves, swamps, and humus, and iron salts.

Odor tests measure the odor produced by the various substances contained in the water, whether vegetable matter in solution, microscopic organisms, or gases of decomposition. Odor is useful in detecting the presence of sewage. The microscopic examination is invaluable for determining causes of odors in surface water.

Turbidity is a measure of suspended matter which obstructs the passage of light. Turbidity may be due to silt, clay, suspended iron salts, organic matter, microorganisms, etc.

Tests of oxygen consumption measure the carbonaceous organic matter which is partly oxidized by acid potassium permanganate.

Nitrogen exists in water in several states of combination. Organic nitrogen, in oxidizing to nitrates, passes through an intermediate stage, the nitrites, or it may be decomposed with evolution of ammonia or free nitrogen. "A state of change is a state of danger."[20e]

Estimation of nitrogen as albuminoid ammonia is an approximate measure of the nitrogenous organic matter. This is from two sources—vegetable and animal. Proteids and amino bodies from vegetable sources are much more stable than those from sewage and evolve nitrogen less rapidly when treated with alkaline potassium permanganate and distilled.

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20. (*a*) Frankland, P., and Frankland, G. C.: Micro-Organisms in Water, London, Longmans, 1894. (*b*) Horrocks, W. H.: An Introduction to the Bacteriological Examination of Water, London, J. & A. Churchill, Ltd., 1901. (*c*) Jordan, E. O., and Burrows, W.: Textbook of Bacteriology, ed. 14, Philadelphia, W. B. Saunders Company, 1945. (*d*) Savage, W. G.: The Bacteriological Examination of Water-Supplies, Philadelphia, The Blakiston Company, 1906. (*e*) Nitrogen in Water, in Prescott, S. C., and Winslow, C. E. A.: Elements of Water Bacteriology, ed. 5, New York, John Wiley & Sons, Inc., 1931.

The presence of nitrogen as free ammonia is a result of decomposition of organic nitrogen, and the test usually indicates roughly the amount of decomposed material. More than 0.15 ppm must be regarded with suspicion.

The presence of nitrites is an indication that either oxidation of organic nitrogen or decomposition of nitrates is taking place. Their presence in any considerable amount must always be regarded with suspicion, although deep well waters of great purity often contain nitrites.

Tests for nitrates measure completely mineralized nitrogen, and their presence in considerable amounts is indicative of past contamination.

Chlorine is found in all natural waters. When the normal chlorine content is known, determination of chlorine is of great sanitary significance. An excess, in absence of soil deposits, is a sure indication of present or past pollution.

Bacteria are valuable indicators of the present condition of any water. The total number is of value in making routine tests from the same source, although liable to insignificant variations. Ordinary procedure enumerates only a part of the bacteria present. Of much more value is the determination of the presence of members of the group of bacteria of which E. coli is the type. They indicate pollution and the possible presence of pathogenic bacteria.

Such is the usual set of standards used by the civil or sanitary engineer in the examination of water.

STANDARD FOR BATHING PLACES

The American Public Health Association [8] has set down certain general principles for bathing-place sanitation. Its committee is of the opinion that all public bathing places, both natural and artificial, should be under the sanitary control of the public-health authorities. The same standards for cleanliness and bacterial purity of the water and the same precautions against the possible spread of disease should apply at both indoor and outdoor swimming pools.

At public bathing beaches on natural waters the same sanitary standards should apply to the bathing houses, dressing rooms, and toilet facilities and to the handling and care of bathing suits, towels, and other articles of bathing apparel as are required at artificial swimming pools.

The Association [8] has laid down rules for the location and the layout of pools, e. g.,

> Coming from the dressing room, a bather should be required to pass the toilets, and go through the shower room before arriving at the pool entrance.

Certain features of design and construction should be incorporated, e. g.,

> In the design of a new pool provision should always be made for complete circulation of water through all parts of the pool during the bathing period.

Also given are the shape, dimensions, depth of water, proportion of deep to shallow water, slope of the bottom and side walls (sloping side walls are dangerous and cannot easily be kept clean), etc.

The Association's handbook suggests a way of proportioning the pool area to the expected load. It states that the consensus of swimming-pool operators was that the space required by a swimmer may fairly be expressed as five-fourths the square of his height and that, on an average, two-thirds of the swimmers present are in the pool at one time. On this basis the average space requirement for an adult swimmer is 36 sq. ft. (3.3 sq. m.).

Outlets should be constructed in such a way that it is possible to drain a pool completely in four hours or less. Inlets for fresh or repurified water should be so located as to produce, so far as possible, uniform circulation of water and the maintenance of a uniform chlorine residual throughout the pool without existence of dead spots.

The Association also specifies the type and care of overflow gutters, steps, runways, and visitors' galleries. For example:

The gallery floor should slope to a drain and should be flushed down with hose regularly. The drainage from the spectator area in no case should be allowed to drain upon the area used exclusively by the bathers. A curb or other provision should be used to prevent litter and dirt from being kicked or scuffed by spectators into the pool or pool area, etc.

Rules for dressing rooms in regard to the type of walls, floors, seats, and lockers, and their proper care and disinfection, are presented, as well as rules for the type and number of showers, toilets, and lavatories. Illustrations are the following: "A bidst or upward flow spray beneath each shower to permit washing between the legs is desirable." "Urinals should be of a type that will not cause splashing of urine upon the legs and feet of bathers."

The handbook specifies under "Recirculation System" that a turnover of pool water in at least eight hours is necessary.

Batteries, of three or more filters arranged in parallel are preferable to a single unit, in order to permit continuation of filtration and recirculation while one unit is out of operation for cleaning or repairs.

Chlorine, the material used to kill bacteria in swimming-pool water, is available in powder, solution, or liquid forms. Once the type of chlorine to use has been chosen, the question of application must be considered. Practically everyone agrees that chlorine should be applied somewhere between the outlet from the pool and the point of return to the pool. Between these two points there will be strainers, filters, heaters, pumps, valves, and perhaps a chemical feed or two. Apparently, opinion is almost equally divided between what might be called prechlorination and postchlorination.

The proponents of prechlorination have many good arguments on their side. Water as it comes from the pool is water that has been used. It contains lint, hair, particles of epithelium from the skin, dirt that has been blown or tracked in, algae, and bacteria. The lint and hair are taken out by the strainers. Between the strainers and the filters it is common practice to add coagulant, alum, and an alkali, such as soda ash. The coagulants react in such a way as to form what is known as floc, a jelly-like curd which entangles the matter too fine to be caught on the screens. The floc and dirt are gathered on the surface of the filter sand, and these are both filtered out of the water, leaving it clear and sparkling.

A large proportion of the bacteria is caught in the floc and is filtered out with it. But the removal of a large portion of bacteria is never enough, because the few organisms left can multiply rapidly and in a short time can become so numerous as to be completely out of control.

Furthermore, some of the bacteria that escape entanglement in the floc on the surface of the sand will become lodged within the bed of sand in the filter. Here, unless killed with an active disinfectant, such as chlorine, they will grow and form lumps which, in turn, entangle minute particles of dirt and even sand itself. These

clumps are known as mudballs, and if left to grow undisturbed they will eventually interfere with the filtering process. The evidence indicates that pretreatment is usually preferable.[21]

When chlorine is applied to water, the residual produced consists of free available chlorine or combined available chlorine (chloramines). Free available chlorine residual is defined as the active form of chlorine present in water solution, as shown when 90% or more of the total ortho-tolidine color develops instantaneously (five seconds), provided the temperature of the sample is not greater than 60 F. Combined available chlorine residual is defined as the combination of chlorine in water solution with nitrogen or one of its compounds. It is popularly known as a chloramine residual. Evidence, however, indicates that free available chlorine residuals have the edge over the chloramine residuals in the treatment of water by virtue of (1) their ability to kill quickly (within three minutes or less), a characteristic highly desirable in a swimming pool, (2) their ability to kill practically all types of algae, and (3) their ability to oxidize iron to a form readily filtrable. The first characteristic ensures a bacteria-free water and the others ensure a clear water. The one stipulation is that the chlorine residual never drop below 0.5 ppm. The upper limit, on the other hand, is on the order of 20 to 30 ppm. When the pH value drops below 7.0, the action of chlorine is greatly accelerated. This acceleration results in a rapid loss of chlorine, so rapid at times that the chlorinator capacity will not be large enough to keep up with the demand. This is one of the reasons for maintaining the pH above 7.0. In fact, there is an increasing tendency to maintain the pH between 7.2 and 7.8. At one time it was the practice to maintain pH between 6.8 and 7.2.

From all available information, the addition of chlorine by use of proper apparatus is today the most satisfactory method of pool disinfection. It is possible not only to disinfect the entire body of water in the pool completely with chlorine, but also to maintain in the pool at all times a residual amount of disinfectant to counteract at once any dangerous pollution disseminated by bathers. With the proper chlorine apparatus it is also possible to increase or diminish the dosage as required to compensate for variations in the bathing load.[8]

The Committee recommends the use of chlorine as a solution of water and either chlorine gas or hypochlorites for disinfection of all pools where there is any appreciable bathing load or where bathing suits are worn.

However, recent developments indicate that the use of ammonia and chlorine for swimming pool disinfection has become increasingly popular because of the more lasting qualities of the *chloramines* thus formed and the possibility of carrying higher disinfecting dosages without production of irritating effects from the use of large amounts of chlorine.

Most experience appears to indicate that better results are obtained by adding chlorine ahead of pool filters.

Intermittent disinfection with hypochlorites as practiced at many pools must be considered a makeshift.[8] It is possible to obtain satisfactory disinfection by intermittent application of these chemicals and to maintain a satisfactory residual chlorine content in the water when the bathing load is constant and not too high. When the bathing load fluctuates widely, the residual chlorine content cannot be adjusted to compensate for the variations, and under excess loads immediate disinfection of infectious matters from bathers may not be accomplished.

21. Griffin, A. E.: Modern Methods of Pool Chlorination, Beach and Pool, August, 1948.

Sterilization of clear water may be obtained by exposure of thin films to ultraviolet rays. There is no conclusive evidence, however, that any residual disinfecting agent remains in the water after such treatment. Until such evidence is produced, it must be assumed that disinfection by this process proceeds according to the law of purification by consecutive dilution and is subject to the limitations imposed by that law. On this assumption there is never any definite and determinable amount of disinfectant in the pool water to act on infectious material which may be discharged by bathers during the bathing period, the time that such material is most dangerous.

Judging from a few reports, a reasonably satisfactory disinfection of water may be accomplished by ozone when the necessary apparatus is properly installed and operated. The data on the use of ozone for swimming-pool disinfection are few and inconclusive.

The Committee of the American Public Health Association recommends the use of copper sulfate in combination with either of the approved methods of disinfection at such times and in such amounts as may be necessary to control growths of algae in swimming pools and other bathing waters. The use of copper salts alone as disinfectants will not produce satisfactory bacterial control and cannot be recommended.

The use of ionized silver for swimming-pool disinfection has been developed abroad and to a limited degree in this country. There is a question of how rapidly this disinfectant acts on infectious material which may be discharged into the pool by bathers. The Committee has not seen fit to recommend the use of ionized silver for swimming-pool disinfection.

Under "Suits, Towels and Caps" the Committee's handbook states that at indoor pools used exclusively by men nude bathing should be required. At indoor pools used exclusively by women, bathing suits should be of the simplest type.

> Suits when used should be of wool or cotton of simple design and of undyed material or tested for fastness of color. Elaborate suits of varied materials or varied color should not be permitted.
>
> At artificial pools all bathers of both sexes should be required to wear rubber bathing caps. . . . All suits and towels must be washed with soap and boiling water, rinsed, and thoroughly dried each time they are used. . . . It is desirable at artificial pools that all suits and towels be supplied and cared for by the management. If individually owned suits are permitted, they should be of prescribed style and material and should be laundered and stored at the pool by the management.[8]

Under "Supervision of Bathers," the handbook includes among other requirements the following: "An attendant should be on duty at the shower room or entrance to the pool to inspect all bathers for skin diseases, open lesions, etc., and to insure that a proper cleansing bath has been taken."

The stipulations under "Personal Regulations" are particularly illuminating:

> A. All persons using a swimming pool must be required to take a cleansing shower bath in the nude, using warm water and soap, and thoroughly rinsing off all soap suds, before entering the pool room or enclosure. A bath after donning suit should not be permitted.
>
> B. A bather leaving the pool room or enclosure for any reason should take a foot bath before returning. A bather leaving pool to use toilet should be required to take a second cleansing bath before returning.
>
> C. All bathers should be instructed to use the toilet and particularly to empty the bladder before taking cleansing bath and entering the pool.
>
> D. Any person having any skin disease, sore or inflamed eyes, cold, nasal, or ear discharges, or any communicable disease, must be excluded from a public swimming pool.

E. Persons having any considerable area of exposed sub-epidermal tissue, open blisters, cuts, etc., should be warned that they are likely to become infected and advised not to use the pool.

F. Spitting, spouting of water, blowing the nose in the pool should be strictly prohibited. Bathers should be instructed that the scum gutter is provided for expectoration.

G. All bathers should be instructed that blowing the nose to remove water is likely to force infectious matter into the sinus and inner ear cavities and possibly cause serious consequences. [The committee should have added, I think, the word "improper" before "blowing the nose to remove water," i. e., improper blowing of the nose by holding one side and causing back pressure could force the water into the sinuses or middle ear cavities.]

H. Divers should be advised to wear rubber caps over the ears or to plug the ears with greased cotton to prevent infection of the ear drum and passages by water forced in by concussion.

Some of the regulations of the Committee listed under "Chemical and Physical Quality of Swimming Pool Water" are as follows:

A. *Excess Chlorine.*—Whenever chlorine, calcium hypochlorite, or other chlorine compounds, without the use of ammonia, are used for swimming pool disinfection, the amount of available or excess chlorine in the water at all times when the pool is in use should not be less than 0.4 p. p. m. or more than 0.6 p. p. m. Whenever chlorine compounds are used with ammonia, the amount of available or excess chlorine should not be less than 0.7 p. p. m. or more than 10 p. p. m.

B. *Acidity; Alkalinity.*—Whenever alum or sulfate of alumina is used during purification or repurification of swimming pool waters, the water at all times when it is in use shall show an alkaline reaction. This means that the hydrogen ion content of the pool water shall not fall below 7.0.

C. *Clearness.*—At all times when the pool is in use the water shall be sufficiently clear to permit a black disc 6 inches in diameter on a white field, when placed on the bottom of the pool at the deepest point, to be clearly visible from the side walls of the pool at all distances up to 10 yards measured from a line drawn across the pool through said disc.

D. *Temperatures.*—The water in any swimming pool should not be artificially heated to a temperature above 76 F. The temperature of the air at any artificially heated swimming pool must not be permitted to become more than 8 degrees warmer than the pool temperature.

Among the things that the Committee stipulates under the heading "Bacterial Quality of Swimming Pool Waters" are these:

A. *Bacteria Count on Standard Nutrient Agar—24 Hours—37 C.—and Confirmed Test.*—Not more than 15% of the samples covering any considerable period of time shall contain more than 200 bacteria per Ml or shall show positive test (confirmed test) in any of five Ml portions of water at times when the pool is in use. All primary fermentation tubes showing gas should be confirmed.

B. The part played by the various strains of streptococci in the intestinal, buccal and nasal discharges makes the presence of streptococci in bathing waters very undesirable. Yet to eliminate them from swimming pools would mean decidedly smaller bathing loads and decided increases in chlorine residuals, either or both of which would hamper the usefulness of the pool.

The Committee calls attention to the fact that streptococci tests are of value in passing on the condition of swimming-pool water, but it does not recommend any uniform standard limit for their presence.

For "Bathing Load Limits" the Committee specifies the following points in the section on frequency of changing water.

1. The purpose of recirculation is primarily twofold, first to remove suspended material and thus produce a clear water, and second, to carry chlorine or chloramine to the water in the pool and thus maintain proper chlorine residuals in the pool water. The clarity of the water is related

to the efficiency of the chlorine, in that the presence of suspended material reduces the effectiveness of the residual chlorine.

2. The total number of bathers using a fill and draw swimming pool shall not exceed one person for each 500 gallons of water in the pool between complete changes of pool water without disinfection, etc.

3. The total number of bathers using an outdoor pool—partly artificial and partly natural in character, dependent for circulation and replenishment upon an inflow and outflow of water from the supply of a clean stream which is relatively free from bacterial pollution or a safe well or spring—might be based upon a figure of one person for each 500 gallons of water added to the pool as a maximum limit, although safe bacteriological conditions should be the principal guide. Where pre-cleansing baths are not used at such pools, reductions in bathing loads will undoubtedly be found necessary to maintain safe bacteriological conditions even with high chlorine residuals. Pre-cleansing baths should, of course, be provided at all pools.

In regard to frequency of disinfection, the Association's handbook points out that experience indicates that far better bacteriological conditions can be maintained with continuous chlorine disinfection.

One bather per 35 to 45 sq. ft. (3.3 to 4.2 sq. m.) of pool area, the handbook notes, will result in safe bacteriological conditions with adequate chlorine residuals.

Under "Outdoor Bathing Places" are considered small streams, lakes, and tidal waters. In a swimming pool the water of which is derived from a public or other supply of unquestioned quality, it may be assumed that the presence of organisms of the coli-aerogenes group indicates pollution by human sewage particles. The presence of such bacteria in outdoor bathing places, however, may be due—as mentioned before—to the wash from cultivated fields, animals, and generally harmless contamination. Routine bacteriological tests do not differentiate between harmful and harmless contamination in such cases.

The use of small, natural, or dammed-up pools along small streams by large numbers of bathers is not recommended unless disinfection is provided.

Where such pools are proposed to be used, and will be dependent upon the natural stream flow for cleansing and dilution, it should be ascertained that there is a constant and appreciable overflow of water past the dam under all weather conditions when the pool is to be used. Any small pool patronized by a number of bathers is certain to show bacteriological pollution in considerable amounts unless disinfection is provided. While no specific amount of diluting water for such pools can be recommended, it is probably fair to say that less than 500 gallons per bather per day is too small a diluting volume without disinfection.

It is recommended that bathing be limited to relatively clear bodies of water and that muddy bottoms which will result in turbid water be avoided.

The disinfection of relatively large bodies of water by use of the so-called chloro-boat has apparently had some success in a few scattered instances. Chlorine and ammonia are undoubtedly more practical of application for large outdoor bathing areas than chlorine alone because of the greater persistence of the chloramines in the water.

Thus, it can be seen that advances in sanitation have kept pace with the rapid increase in public bathing places throughout this country. Sanitarians now give assurance that they can kill or neutralize 99% of the bacteria in a pool. "Can kill" should be emphasized, for in many states and cities the laws are so recent that the old pools and bathing areas have not been brought up to the present standards. Only newly built pools or new bathing areas are required to meet the acceptable standards. Thus, even with modern setups there are many bathing places today that

fall far below the standards set down in 1942 by the American Public Health Association.[22]

SKIN AND WATER

Even though bacterial contamination of the water can be controlled, it remains that the skin of the body cannot stand too long the vitiating effect even of pure water.

Normal skin acts as a barrier to micro-organisms, but the glandular orifices present vulnerable points, and infection usually enters the skin at a glandular orifice, or through a break due to injury. Further protection is afforded by the normally acid reaction of the skin (pH 5.5) which is apt to retard the growth of micro-organisms.[23]

According to Linksz [24]:

The surface of the skin is protected and lubricated by a thin waterproof coating, the product of the sebaceous glands, the sebum. Under normal conditions the emulsion of moisture (sweat) and fat (sebum) on the surface of the skin is such as to secure proper turgescence and greasiness, maintaining adequate perspiration at the same time. If this proper emulsion becomes disturbed, the perspiration increases, the skin loses its proper humidity and becomes either "rough" if the fat is scarce or "oily" if there is an overabundance of nonemulsified fat. The fatty acids produced by the skin are those natural antibiotics on which the organism can rely to such amazing extent to keep the ubiquitous bacteria from penetrating into deeper structures.

Linksz went on to say:

Exaggerated cleansing deprives the skin of its protective film; so do compresses. Peculiar as it may sound, water, by disturbing the physiologic emulsion of moisture with fat on the surface of the skin, desiccates the skin and favors its withering. . . . When water or very diluted (hypotonic) solutions come in contact with tissues, the cells absorb fluid, resulting in swelling, even rupturing of the cells. If the solution is highly concentrated (hypertonic), fluid from the cells diffuses into the solution and causes shrinkage of the cells.[24]

Stokes stated that use of "grease or a detergent substitute to restore the fat-protection against infection and allergens, and the preservation of the 'acid mantle' by avoiding the raising of the skin pH by any agent" are important.[25]

MAN'S LACK OF AQUATIC ADAPTATION

Another factor is man's lack of adaptation to an aquatic environment. Taylor, of Jacksonville, Fla., has given us a clear picture of this reason for man's being subject to otitis and sinusitis from swimming.[26] He stated that he had seen patients with infection of the ears or sinuses who had frequented a swimming pool

22. Wallace and Tiernan Products, Inc., Belleville 9, N. J.: Personal communication to the author, June, 1949. Rules and Regulations of the Department of Health of the Commonwealth of Pennsylvania for the Prevention of Diseases and for the Protection of the Lives and Health of the People of the Commonwealth to Carry out the "Public Bathing Law" of June 23, 1931. Elias, George A., head of State of Pennsylvania Health Engineering Department: Personal communication to the author, April, 1949. Act of General Assembly No. 299, Providing for the Regulation of Bathing Places, Swimming Pools, and Natatoriums, Pennsylvania, 1940. Act No. 143 on Bathing Places in Pennsylvania, May 1, 1945.

23. Becker, S. W., and Obermayer, M. E.: Modern Dermatology and Syphilology, Philadelphia, J. B. Lippincott Company, 1940, p. 11.

24. Linksz, A.: Course 163, American Academy of Ophthalmology and Otolaryngology, October, 1949.

25. Stokes, J. H.: "Simplifying" Dermatology for the Practitioner, Pennsylvania M. J. **52**:1065, 1949.

26. Taylor, H. M.: Otitis and Sinusitis in the Swimmer, J. A. M. A. **113**:891 (Sept. 2) 1939.

fed by a spring having a flow in excess of 145,000 gallons a minute while others had used exclusively a pool with a flow in excess of 365,000 gallons a minute. Chemicals that often lead to irritation of the nasal mucous membranes were not used in either of these pools because of the low bacterial counts found in repeated examinations of the water by the Florida State Board of Health. . . . The water showed no evidence of contamination [and could be considered] relatively pure spring water having a temperature of 71 F., and their use of these bathing places, with their large amount of water supply and its relative degree of purity, served to emphasize the significant role of factors other than contaminated water in the etiology of infections of the ear or sinuses associated with swimming. . . . . .

A cogent factor in the etiology of infections of the upper part of the respiratory tract associated with swimming is man's invasion of an environment to which he is not adapted. . . . He is a terrestrial being, adapted to his environment, but he is without important structural adaptations for an aquatic environment. This lack of anatomic and physiologic modifications is in contrast to the presence of structural adaptations in the animals whose normal habitat is water.

In all the air-breathing animals, including man, the usual stratified flat epithelium lines the nasal vestibule and gradually assumes the structure of the stratiform ciliated cylindric epithelium, the true respiratory mucous membrane. This ciliated epithelium warms the air and keeps moist the tracts through which a current of air is constantly passing. It is designed also to secrete a mucus, both bactericidal and inhibtory to bacterial growth, that enmeshes the bacteria and carries them by a wavelike motion to the pharynx and esophagus for disposition. The difference between the upper part of the respiratory tract of man and that of the aquatic animals lies not in the mucous membrane but in the ability of animals highly modified for life in the water to close involuntarily or at will the anterior nares, an adaptation conspicuously lacking in man. They are thus able to protect the respiratory epithelium from the destructive action of water.

In every species whose normal habitat is water, some provision for excluding water from contact with the respiratory mucous membrane is found, whether in reptilian, avian or mammalian life. The alligator (Alligator) represents the highest form of reptilian life. The muscles of the anterior nares of this aquatic reptile are so arranged that they close the nostrils when the head is submerged. There is a wide variation in the means by which aquatic birds protect the respiratory mucous membrane from water. The pelican (Pelecanus), the cormorant (Phalacrocorax), the snake bird (Auhinga) and the booby gannet (Sula) have no external nares. The diving petrel (Hydrobatida) has external nares, but they may be closed by an arrangement similar to that in the alligator. The frigate bird (Fregata) has a scalelike membrane entirely closing the external nares. Though the external nares of some of the aquatic birds cannot be closed, water is not allowed to come in contact with the part of the respiratory passage that is lined by respiratory mucous membrane.

The most consistent modification of aquatic mammals for life in the water is the ability to close the external nostrils. In the Hippopotamidae, according to Howell,[27] the nostrils take the form of a pair of slits, closure seems to be partly voluntary and opening is accomplished by muscular pull along both borders of the slits. Closure is accomplished in various ways, and probably no two species have exactly the same mechanism for attaining this end. . . .

Another consistent modification that all aquatic animals have for life in the water is the ability to close the ear in order to prevent water from coming in contact with the membrana tympani. In view of this provision, in man the lack of ability to close the external ear is but another evidence of the fact that he is a terrestrial animal and does not have the modifications necessary for life in the water. As Howell[27] observed, it is doubtful whether any aquatic animal swims with the external auditory canals open, and when the head is submerged he is dependent entirely on bone conduction for his hearing. The alligator (Alligator) has, immediately behind the eye, a hinged, cornified flap that constitutes a covering for the ear. It closes tightly as the reptile submerges. The earless seal (Phoca) has no pinna, and the external opening of the external auditory canal is so tightly closed that the lumen can scarcely

27. Howell, A. B.: Aquatic Mammals, Their Adaptations to Life in the Water, Springfield, Ill., Charles C Thomas, Publisher, 1930, pp. 87 and 68.

be discerned. After recognizing these facts pertaining to the adaptations that aquatic animals have for protecting the drum membrane against water and realizing man's lack of like modifications, one can hardly escape the conclusion that a perforation of the drum membrane is in man a positive contraindication for swimming or diving.

Otitis externa, or furunculosis, from which the swimmer often suffers, likewise illustrates that the external auditory canal was not intended to withstand the vitiating effect of water. If the hand is immersed in sterile water for thirty minutes, the skin will be wrinkled and degeneration of the epithelium will result. A similar change takes place in the ear of the swimmer or diver. Maceration of the delicate dermis by the water breaks the skin and opens up for the ever present staphylococcus an avenue of infection, with resulting furunculosis.

LOSS OF BODY HEAT

Still another factor related to aural and sinus infections, according to Taylor and Dyrenforth, is that of chilling of the body surfaces.[28] They state that the human organism must maintain a constant average temperature of 98.6 F., for any considerable deviation from the average for appreciable periods will result in morbid changes.

Cold water has a veritable appetite for heat. Since the ratio of conductivity of water to air is 27 to 1, it follows that water takes heat from the body twenty-seven times faster than does air. The lack of a compensating mechanism for the maintenance of an average normal temperature in any medium colder than his normal surroundings is conspicuous in man. Many arctic birds maintain a normal temperature above 102 F. (38.9 C.) throughout continued periods of exposure to icy waters, as do the arctic Mammalia, yet man's loss of body heat during submersion for only fifteen or twenty minutes in water at a temperature of 70 F. (21.1 C.) may be five times the normal basal rate. Mudd, Goldman and Grant[29] reported the striking effect of this loss of body heat on the nasal mucous membranes. In their experiments, carried out by placing in the nares a sensitive instrument devised for registering temperature, they recorded depressions as great as 10.8 degrees F. (6 degrees C.), their observations indicating the great amount of vasoconstrictor response in this area.

Taylor states[30] that he observed the effect of chilling on 250 children under 13 yr. of age by taking their temperatures before and after a period of 45 min. devoted to swimming in an indoor pool with the temperature of the water at 73 F. In only 30 of this number was a normal temperature maintained; in many there occurred a drop to a low of 95 F.

Bacteria are normally and constantly present in the upper respiratory passages, but they may multiply to pathologic proportion in the person whose resistance is lowered.

Kuntz[31] made clear the interrelation of the innervation of the peripheral blood vessels and the mucous membranes. This anatomic control of vasoconstriction and vasodilation forms the basis of the physiologic balance between the splanchnic and the peripheral areas.

Thus, when stimulation by cooling results in peripheral ischemia, the mucous membranes undergo a similar experience; prolongation of this condition serves to throw out of balance the nicety of integration existing under normal metabolic

28. Taylor, H. M., and Dyrenforth, L. Y.: Chilling of the Body Surfaces, J. A. M. A. **111**:1744 (Nov. 5) 1938.

29. Mudd, S.; Goldman, A., and Grant, S. B.: Reactions of the Nasal Cavity and Postnasal Space to Chilling of the Body Surfaces, J. Exper. Med. **34**:11, 1921.

30. Taylor, H. M.: Sinusitis and Swimming, J. A. M. A. **85**:7 (July 4) 1925.

31. Kuntz, A.: The Autonomic Nervous System in Relation to Otolaryngology, J. A. M. A. **107**:334 (Aug. 7) 1936.

phases and accounts for the ability of ordinary avirulent organisms to overcome the obstacles of tissue immunity at the portals of entry.

In Taylor's experimental studies the most important single factor regarding the establishment of infection is that during exposure peripheral vasoconstriction is prevented by muscular activity, and it follows that infection is, therefore, less likely to occur. The conclusions then were that chilling without exercise produces a leucopenia of the polymorphonuclear neutrophilic type. Taylor reported in 1936 [32] the following observations on a group of swimmers:

> Eight of the lifeguards [at Jacksonville Beach, Fla.] were subjected to forty-five minutes of swimming in ocean water at a temperature of 68.5 F. The weight, tempe-ature, blood pressure and blood count of each swimmer were ascertained prior to and immediately following the period of swimming. After the swim, all the participants exhibited a diffuse generalized purplish hue. Their nail beds were moderately cyanotic; likewise their lips. The rectal temperature showed an average reduction of 4 degrees. The erythrocyte count showed an increase of from 700,000 to 1,500,000. The leukocyte count showed a consistent increase of from 8,000 to 10,000 white cells.

In another experiment of this kind there was a relative leucopenia, in regard to both the total number of white cells and the polymorphonuclear neutrophil count.

Taylor's conclusions were as follows: Chilling of the body surfaces without compensation leads to morbid changes within the body. They are caused (1) by peripheral vasoconstriction with attendant peripheral stasis and anoxemia, (2) by lowered leucocyte response, both total and polymorphonuclear, and (3) by impairment of the phagocytic capabilities of the fixed tissue cells, including that of the nasal mucous membrane. One of the immediate results of these changes is the predisposition to infections of the upper part of the respiratory tract, the paranasal sinuses, the Eustachian tubes, and the middle ear.

It is interesting to note that irritation of the body surfaces may come from still another source, that of the variable pH of different bodies of water. Swimming-pool water is now optimally adjusted to a pH range of 7.2 to 7.8.[21] As mentioned previously, it was a former practice to maintain a pH of 6.8 to 7.2; the change was made because the action of chlorine is greatly accelerated below pH 7.0, which results in too rapid a loss of chlorine. It is illuminating to contrast this water pH with the pH of different body surfaces: that of the skin is nearly 5.5; that of the eye is from 7.3 [24] to 8.0; [33] that of the nose, 7.56.[34] Thus, the pH range for the eye seems to fit into the latest pH range used for swimming pools. In fact, Gifford [33] has said that isotonic sodium chloride in a pH range of 8 to 7 causes no sensation or irritation in the eye. However, above or below this level the eye is annoyed. And this is for isotonic sodium chloride solution! It was mentioned previously that the skin pH (5.5) should not be raised, as this destroys the protective acid mantle of the skin.[23] The pH of the nose is not far from the range now used for swimming pools. However, in how many places in the world where there is swimming is there someone to adjust the pH of the water every few hours?

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32. Taylor, H. M., and others: Report of Committee on Otorhinologic Hygiene of Swimming, Tr. Sect. Laryng. Otol. & Rhin. of the A. M. A., 1936, p. 300.

33. Gifford, S. R.: Hand-Book of Ocular Therapeutics, Philadelphia, Lea & Febiger, 1942, p. 6.

34. Buhrmester, C.: A Study of the Hydrogen-Ion Concentration, Nitrogen Content, and Viscosity of the Nasal Secretion, Ann. Otol. Rhin. & Laryng. **42**:104, 1933.

Thus, so far seven factors have been mentioned that can lead to infection of the ears from contact with water as in swimming:

1. Pure water itself can rob the skin of its protecting mantle of fatty acids.
2. Pure water can raise the pH of the skin from the proper acid level of 5.5.
3. Chemicals used in the water for purification may act as irritants.[26]
4. Bacterial or mold contamination in the water can cause infection.
5. There is lack of adaptation of the human ear in its failure to have a closing flap to keep the water out.
6. Chilling of the body surfaces lowers the resistance to organisms that might be already present in the ear.
7. Bacteria and molds are present in the ear canal in either normal or abnormal amounts.

Linksz [24] speaks of the "seborrheic habitus," in which the fatty acids in the skin are apparently diminished and changed and the yeasts and bacteria are present in greatly increased numbers. Gill [35] stated in connection with molds, "Swimming is held responsible for infection in many cases. Probably the only relationship in most instances is the supplying of the necessary moisture that enables molds to grow prolifically."

Sea water, which is 3% salt, is even more caustic on the delicate eardrum membrane than fresh water. In the Hawaiian Islands exostoses of the ear canal are frequent in the pearl divers because of the constant irritation of the water. In fact, Fowler and Osmun [36] found that cold water itself (67.5 F.) can produce hyperostosis and exostoses of the external auditory canal.[37]

Beard and Meadowcroft [38] stated that the results of their experiments indicate that there is a difference in the death rate of typhoid organisms in filtered sea water as compared with unfiltered sea water. The death rate was much higher in the unfiltered sample, although organisms were recovered on the ninth day even in the unfiltered water. They were recovered as late as the 14th day in the filtered water.

The above reference shows clearly that salt water, like fresh water, may act as a culture medium for pathogenic bacteria.[39]

PREVENTION AND TREATMENT OF OTITIS

What, then, is to be done about this problem?

I have stated above that a proper method of breathing is one way of keeping water from coming in contact with the nasal mucous membranes and from thence with the middle ear. The nasal clip is the second method.

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35. Gill, W. D.: Diseases of the External Ear, in Jackson, C., and Jackson, C. L.: Diseases of the Nose, Throat and Ear, Philadelphia, W. B. Saunders Company, 1945, p. 245.

36. Fowler, E. P., Jr., and Osmun, P. M.: New Bone Growth Due to Cold Water in the Ear, Arch. Otolaryng. **36**:455 (Oct.) 1942.

37. Gilse, P. H. G.: Des observations ultérieures sur la genèse des exostoses du conduit externe par l'irritation d'eau froide, Acta oto-laryng. **26**:343, 1938.

38. Beard, P. J., and Meadowcroft, N. F.: Survival and Rate of Death of Intestinal Bacteria in Sea Water, Am. J. Pub. Health **25**:1023, 1935.

39. Elder, F. B., of the American Public Health Association: Personal communication to the author, December, 1949.

Skinner gives this advice in *Hygeia*:[40] "Let the body be in the water, but keep the head in the air." Good advice, but not often followed!

A better measure would be use of a water-repellent plug that easily conforms to the shape of the ear canal, large or small, straight or tortuous; a plug that can be put in fresh each time and that has some antiseptic value should a drop of water inadvertently slip into the ear canal or should some bacteria or debris be pushed further into the canal by the plug. Such a combination of factors would be met by an oily ear plug, a tight oily plug with antiseptic properties. Oil protects the skin from the macerating effects of the water and is also water-repellent.

Thus, if a tight plug was made of an oily material that could be easily applied and just as easily removed from the ear canal, and if it had bactericidal properties, should a drop of water enter the canal, this would be ideal for our purpose.

Chlorine is one of the time-honored bactericides for the treatment of water; in fact, even in the field of antisepsis and wound treatment its use antedates the Listerian era. Remington[41] stated, "It does not precipitate proteins, but on the contrary dissolves necrotic tissue which thus helps to keep a wound clean." Chlorine is effective against various types of pathogenic micro-organisms that might be in or enter the ear canal, Gram-positive and Gram-negative, aerobic and anaerobic bacteria, and also against fungi, yeasts, and Protozoa. Chlorine may be used repeatedly without the risk of development of drug-resistant organisms or the likelihood of sensitization. One would not dare to use the antibiotics or sulfonamides repeatedly. Even the mercurial antiseptics have their quota of patients in whom a sensitivity to their use develops.

The problem, then, is to find for this relatively ideal bactericide a bland, nonirritating, stable solvent that is oily and yet allows the chlorine to ionize in water.

It has been the practice in one of this hospital's clinics,[42] for many years now, to use a pack saturated with 25% *m*-cresylacetate in chlorinated paraffin (chlorcosane) for the treatment of otomycosis. Chlorcosane is a light paraffin oil which has been treated with chlorine to the point of saturation. *m*-Cresylacetate (cresatin®), a colorless oily liquid, is the acetic acid ester of *m*-cresol. It has been used alone or in combination with thymol by many otologists for the treatment of fungous infections of the ear canal. The volatility of *m*-cresylacetate also seems to facilitate contact of the medicament with areas of potential infectivity other than the apparent site of application, since it has been established in vitro that *m*-cresylacetate will inhibit the growth of mycelia and spores at some distance from the point of application. This property can have prophylactic, as well as therapeutic, implications. Here would seem to be the ideal combination for the present purpose, the oily fungicide in an oily chlorinated base. However, the chlorine in the chlorinated paraffin was not ionizable in water.[43] This was disappointing, but, after a thorough

40. Skinner, G. A.: Advice to Swimmers, Hygeia **17**:549 (July) 1935.

41. Remington, P.: Practice of Pharmacy, ed. 8, edited by E. F. Cook and C. H. La Wall, Philadelphia, J. B. Lippincott Company, 1936, pp. 584-909.

42. Watt, R. W., of the Ear, Nose and Throat Service of Frankford Hospital: Personal communication to the author, 1941.

43. (*a*) Josselyn, L. E., Abbott Research Laboratories: Personal communication to the author, Jan. 12, 1949. (*b*) Goodman, L., and Gilman, A.: The Pharmacological Basis of Therapeutics, New York, The Macmillan Company, 1941, pp. 848-849.

search of the literature and textbooks, a better compound was found in the form of chloroazodin U. S. P. (azochloramid®) dissolved in glyceryl triacetate (triacetin). This proved to be a truly remarkable base for our purpose. Glyceryl triacetate is a bland, virtually nonirritating oily solvent which is quite stable. The most remarkable feature is that it is both fat and water-soluble. Thus, oily *m*-cresylacetate can be dissolved in it; yet, if water comes into contact with the solution, the chloroazodin in glyceryl triacetate is liberated to go into solution in the water and thus free its chlorine to any organic matter present. An additional advantage is its low surface tension, which further aids in the penetration of crevices inaccessible to aqueous solutions. Furthermore, the oily nature of the solvent is of great value in preventing irritation of the skin in cases requiring prolonged application.

Chloroazodin solutions themselves—in glyceryl triacetate or in water—are distinguished from solutions of other chlorine bactericides by their unusual stability and their gradual liberation of chlorine over long periods, even in the presence of such organic matter as serum and exudate.[43b] Because of the relatively small amount of free chlorine present at any one time, chloroazodin solutions in therapeutic concentrations are nontoxic to tissues and are virtually nonirritating.[44] In addition, chloroazodin solutions are nonstaining and odorless. Chloroazodin is sensitive to light and excessive heat. However, if stored in dark brown bottles in a cool place, solutions retain their potency for long periods—a great advantage over other chloramine-type bactericides.

The *m*-cresylacetate was used in a 25% solution in the chloroazodin because when applied in a higher concentration it had occasionally caused some irritation, probably from its mild keratolytic effect. Thus, the solution used was one part of *m*-cresylacetate to three parts of chloroazodin in glyceryl triacetate. (The chloroazodin, if preferred, may be incorporated in an olive-oil base.)

A comfortable and efficient ear plug which conforms to the shape of any ear canal can be made up fresh each time by the patient from nonabsorbent cotton. This is a form of cotton which has been purified, but from which the oil has not been removed.[44] Its special use is in the bacteriological laboratory for stoppering flasks or test tubes containing liquids to be sterilized. It is water-repellent and forms an efficient, nontraumatic cork in the ear canal against the entrance of water. If about four drops of the *m*-cresylacetate-chloroazodin solution is placed into each ear and a tight plug of nonabsorbent cotton is inserted, the result is both a protection against water coming into contact with the canal or middle ear and an oily antiseptic which guards the skin and tends to kill any offending organism that might be activated by the water or brought into the ear in the water.

Although staphylococci, diphtheroids, sarcinae and micrococci are found in normal aural canals,[45] it is possible for them to act as etiologic agents in ears where infection may be precipitated by decrease of local resistance and trauma.[46]

This lowering of local resistance in the ear canals can be brought on by water while in swimming.[6] Thus, the antiseptic ear plugs can prevent infection that is

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44. Lederer, H. G. (bacteriologist): Personal communication to the author, June, 1949.

45. Syverton, J. T.; Hess, W. R., and Krafchuk, J.: Otitis Externa: Clinical Observations and Microbiologic Flora, Arch. Otolaryng. **43**:213 (March) 1946.

46. Conley, J. J.: Evaluation of Fungous Disease of the External Auditory Canal, Arch. Otolaryng. **47**:721 (June) 1948.

water-induced as well as water-borne. The oily antiseptic coats the ear canal, and the water-repellent plug closes the canal against the entrance of water. Should a drop of water get through, the chlorine becomes dissolved in it and makes it sterile.[47]

ILLUSTRATIVE CASE REPORTS

CASE 1.—E. L., a 35-yr.-old woman, was seen on Aug. 1, 1947, with discharge from both ears and large central perforations in each tympanic membrane. The ears had been troublesome since an attack of scarlet fever at the age of 7 yr. The ears were treated and finally made dry, only to have the discharge return again after swimming in an open-air pool where intermittent chlorination was used. This happened a number of times, necessitating repeated treatment. She was advised that special treatment might eventually close the perforation in her eardrum membranes and render her ears less susceptible to trouble from swimming. However, in the meantime, she would have to have some protection. The nonabsorbent-cotton ear plugs and a solution of one part of *m*-cresylacetate to three parts of chloroazodin in glyceryl triacetate (1 : 500) were prescribed for use in swimming. The eardrum membranes were finally healed without any further delay from recurring discharge.

CASE 2.—N. P. was a 40-yr.-old man working for a graduate degree and living at a New Jersey sea-coast resort while writing his thesis. He had spent most of his summers at this resort since a small child and was a great swimmer. His winters latterly were spent inland in a college town, where he frequented the school swimming pool almost every day. His chief complaint on June 30, 1949, was that both ears were sore, and his local physician had told him to stay out of the water permanently because of exostoses in both ear canals. The ears were treated, and the nonabsorbent-cotton ear plug was prescribed, along with a solution of one part *m*-cresylacetate to three parts of chloroazodin. He had no more trouble.

CASE 3.—J. B. was a 30-yr.-old woman seen first on Feb. 2, 1947, for recurring symptoms of labyrinthitis. A radical mastoidectomy was done, with the Portmann type plastic operation to the ear canal. After she was fully recovered, she expressed a desire to go swimming as had been her habit before her illness. This was advised against, but, sensing that the patient might go anyway, the nonabsorbent-cotton plugs with the solution of two parts of *m*-cresylacetate and two parts of chloroazodin in glyceryl triacetate (1 : 500), were prescribed. She went swimming with no untoward sequelae.

CASE 4.—J. P., a 16-yr.-old boy, was seen on July 2, 1949, in the outpatient department of the Episcopal Hospital of the University of Pennsylvania with acute external otitis of mixed type—fungus and bacterial. He had been swimming and wanted to go again over the coming holiday. He was administered 7.7 grains (0.5 gm.) of sulfadiazine every three hours and given a prescription for the nonabsorbent-cotton plugs with a solution of two parts of *m*-cresylacetate and two parts of chloroazodin in glyceryl triacetate (1 : 500). He was told that he might go swimming, which he did. When he returned to dispensary the next week after using the liquid, both as a treatment (1 drop every three hours) and for swimming, his ear infection was practically cleared.

CASE 5.—M. E. was a 23-yr.-old woman with a perforated eardrum membrane and recurring ear discharge every time she went swimming or to the hairdresser. This delayed treatment for closure of the perforation until use of the nonabsorbent-cotton plugs with a solution of one part *m*-cresylacetate and three parts chloroazodin in glyceryl triacetate (1 : 500) were prescribed.

CASE 6.—D. C., a 27-yr.-old man, had recurrent otomycosis from swimming or bathing. He was given the nonabsorbent-cotton plugs and solution of two parts of *m*-cresylacetate with two parts chloroazodin in glyceryl triacetate (1 : 500). There was no further trouble.

It is to be noted in these case reports that the solution used was varied from one to two parts of *m*-cresylacetate. One part *m*-cresylacetate was used in cases of

47. Kembring, R. (research chemist): Personal communication to the author, June, 1949. Kennedy, R. H.: Use of Azochloramid in Infected Wounds, Am. J. Surg. **31**:294, 1936. Young, F.: The Treatment of Surgical Infections with New Chlorine Solutions, Surg., Gynec. & Obst. **63**:318, 1936.

a mild nature or when the solution was to be used frequently for swimming over short periods of time. As *m*-cresylacetate is somewhat irritating to some skins, this was thought advisable. Two parts *m*-cresylacetate were used in the severer infections or when the solution was not to be used too often. The selection at times was somewhat arbitrary.

CONCLUSION

The material used in the ear plugs recommended is cheap, comfortable, and disposable. It obviates difficulties with the small percentage of persons who are sensitive to wool—such as with the use of wool plugs. Gill wrote, "Ear stoppers do no good unless their use is followed by some antiseptic in the canal." He advocated the use of mercury cyanide (1 : 5,000) in 70% ethyl alcohol on removal of the ear plugs after swimming.[48] However, with the *m*-cresylacetate-chloroazodin combination, the antiseptic is present from the time the plug is inserted. The oil protects the skin of the ear canal from the loss of its fatty acids and helps it retain its normal pH by the oil's water-repellent character. *m*-Cresylacetate not only is a fungicide but has been used widely for the treatment of furunculosis,[49] which is a common result after swimming. In fact, the *m*-cresylacetate-chloroazodin solution has been in constant use in my office for some time for the treatment of bacterial and fungous type ear infections. The tight warm plugs protect the ear canal from contact with cold water and its irritating effects.

48. Gill,[35] pp. 255-266; Lecture Course on Diseases of the External Ear, American Academy of Ophthalmology and Otolaryngology, October, 1949.

49. Gill.[35] Lederer, F. L.: Diseases of the Ear, Nose and Throat, Philadelphia, F. A. Davis Company, 1938, p. 109.

THE FUTURE OF OTOLARYNGOLOGY

President's Address

SHERMAN L. SHAPIRO, M.D.
CHICAGO

IT IS twenty-six years since I had my introduction to otolaryngology as intern, or middler, in the Ear, Nose and Throat Ward of the Cook County Hospital. There have been changes in all branches of medicine since that time, but none, it seems to me, have been as great as those which have taken place in our field. Although much of the following is doubtless familiar to you, especially to the older men, it may be helpful to evoke for a little while a picture of otolaryngology as seen through the eyes of a beginner in 1925.

As middler on the service it was my job to care for patients after frontal sinus operations, and there were always two or three such patients around. The operation done was a Killian or a modification by Skillern with a rubber catheter for irrigation projecting from the brow and the nose. The same operation was done for either acute or chronic sinusitis. The patients generally got well, some died, and convalescence was always long, often with sequestration. Not until a few years later did we learn that a small trephine opening was best for acute frontal sinusitis.

In the outpatient department there were always several patients with atrophic rhinitis and ozena to be treated with scarlet-red emulsion or packings of tampons of ichthammol (ichthyol®) in glycerin. Many of the patients were anemic-looking young women. I wonder what has happened to these people! They seemed to disappear from both clinic and private practice a few years later, just about the time vitamins came into general use.

Twenty-six years ago certain otolaryngologists such as Dr. Pierce and Dr. Sonnenschein were noted for their skill in the refinements of office practice and for the elegance of their prescriptions, which were eagerly sought after by neophytes such as I. Other men carried this elegance of perscriptions to greater lengths, using as many as six different kinds of aromatic oils with which they sprayed their patients' noses. Perhaps it was one of these who inspired the familiar wisecrack of Dr. Church, a crusty neurologist of this period, that another name for an otolaryngologist should be "psst, psst, come back Thursday."

The first Western Electric Audiometer—a large Model 1A—was gladly loaned without charge to the Ear, Nose and Throat Department of the Cook County Hospital and tried by us a few times when we had nothing else to do. It was agreed by practically everyone that it was just another unnecessary gadget.

Endoscopy was done by anyone who wished to with Jackson, Brunings, Mosher, or Lynch Suspension instruments. The latter were a favorite with Dr. George Boot for intralaryngeal surgery. Aside from the fact that general anesthesia was required.

Read before the Chicago Laryngological and Otological Society, Chicago, April 23, 1951.

the Lynch outfit was perfect for this purpose but never achieved any popularity, possibly because Dr. Lynch, a great but extremely modest laryngologist, never bothered to broadcast the merit of his instruments.

In 1925 many otolaryngologists in Chicago refused to see patients by appointment; some handled as many as 90 or 100 patients a day in their office. In 1925 also, the Public Health Institute, founded the previous year and backed by Dr. Louis Schmidt, was enlarging its activities, and our profession was feeling the first chill breath of socialized medicine. It was about this time that an attending man came in one day with a sample of a new alkaloid extracted from the recently discovered Chinese plant mahuang, which was said to have a more prolonged action on the nasal mucosa than epinephrine. This incident marked the initial appearance of what is probably the most widely used nasal medication today. As a footnote to the rise of ephedrine can also be recorded the end of another prescription for stuffy noses which was highly favored by certain rhinologists—cocaine solutions in oil. These were dispensed as Dr. So and So's nose drops and were very popular with the patients, providing not only relief for the nose but also a pleasant jag.

It was around this time, too, that one of my attending men by the name of Dr. Sam Salinger decided to make nasal plastic surgery respectable. This type of surgery had previously been the property of two different types of physicians. In Chicago it belonged to the quacks, some of whom bandaged the eyes of their patients so that the latter could not see who was operating and thus could not sue in case of a bad result. In New York and abroad there were a few first-class men, such as the great Dr. Joseph, who either refused to teach any one their art or charged physicians large sums for watching them but worked in such a manner that one could see very little. As an intern in his service, it was my privilege to watch Dr. Salinger overcome these handicaps to become an acknowledged master in this field.

I cannot remember a single mention of the word allergy twenty-six years ago, but I do recall that a former resident boasted of having done one hundred mastoidectomies over a six-month period. There were always patients with sinus thrombosis, brain abscess, or otitic meningitis around the ward. Physicians operated with the skill and assurance born of constant practice. It was only a couple of years later that Lempert published a description of his endaural mastoid operation. The procedure was shrugged off by the great otologists both here and abroad as a foolish and unnecessary refinement.

It might be of interest to continue these reminiscences, but I believe enough has been said to illustrate my point. Let me state, however, that my remarks are not offered in any spirit of criticism and certainly not with any feeling of nostalgia. The advances in our field since 1925, especially those connected with chemotherapy, have been truly wonderful for the patient, who is, after all, our chief concern. There are some compensations for the otolaryngologist too. If the antibiotics have placed a strain upon our pocketbooks, it is fair to state that they have lifted one from our coronary arteries. Few physicians who have ever dealt with a fatal ear or sinus complication would, in my opinion, be willing to go back to the old days.

There are, of course, factors besides the antibiotics which account for the reduction in surgery. Even before chemotherapy made its appearance, clinical experience as well as the researches of Proetz and other students of nasal physiology had begun to deflate the value of surgery in chronic suppurations of the nasal sinuses; this tendency was accelerated as we became aware of the great role played by allergy

in the production of nasal symptoms. These and similar advances were generally welcomed by otolaryngologists. Not so welcome were other changes which were due simply to the encroachment of physicians from other fields into what was formerly our own domain. We know that most of the tonsils that are removed today are removed by general physicians or surgeons; we have heard long ago of the "pediatric ear," and we are aware of the extent to which endoscopy is practiced by chest surgeons and specialists in internal medicine.

The changes I have mentioned, both welcome and unwelcome, which have eliminated a considerable amount of work formerly done by otolaryngologists, have not for various reasons seriously affected the older men in our field. They do, however, pose the problem of the future of our specialty. If young men of high caliber are to be attracted to otolaryngology, they must be assured of a livelihood. This cannot be done by throwing a fence around any procedures in our field but must be accomplished by expanding the latter to include a number of conditions which we formerly neglected. Men like Dr. French Hansel, of St. Louis, in allergy and our own Dr. Sam Salinger in plastic surgery have shown the right way. I believe that allergy should be a compulsory instead of a voluntary part of resident training if we are to achieve high standards of competence in this field. This may be done by cooperation with allergy departments, but a better way is probaby for otolaryngology departments of teaching institutions to teach allergy themselves. There are sound reasons for such an arrangement other than those related to resident training. The diagnosis and treatment of allergic conditions in the department of otolaryngology would make for a better correlation in the clinic, would help to fix allergy as a part of our specialty, and, finally, would give us a chance to evaluate fresh ideas by otolaryngologists who are working in this field. This is all the more important since we all know that there are many cases of hyperesthetic rhinitis and similar conditions which cannot be helped by conventional allergic methods.

In the domain of nasal plastic surgery we can well be proud of the type of work being done by those otolaryngologists who have gone into this field. I see no reason why this should not be extended in time to other plastic procedures on the head and neck. Another field which needs more of our attention is that involving injuries of the head and face. In the former, modern vestibular tests are, in my experience, a far better means of evaluating chronic disability than the usual examination by a neurologist; in the latter, the treatment of fractures and other injuries about the face is something which the otolaryngologist can readily learn. Other maxillofacial conditions, including cleft-palate work, have been done by men in our field before and can be done again.

I am aware that the American Academy of Ophthalmology and Otolaryngolgy has had didactic courses in these borderline subjects, but more than that is needed. There are many difficulties, of course. Few heads of departments of otolaryngology are as fortunate as is Dr. Lierle at the State University of Iowa, who has both oral surgery and otolaryngology under his jurisdiction and can give his residents a very broad training. Possibly some of these difficulties can be solved by exchange residencies or preceptorships. However, in spite of difficulties, progress is being made in Chicago toward planning the future of our specialty. In this connection credit must be given to one of our members, Dr. Lederer, of the University of Illinois, who, to my personal knowledge, has gone to considerable trouble to attract men with

training in border-line subjects to his department. Both his residents and those at the University of Chicago have the option of taking an additional two years' training with the surgery department. You heard one of these residents from the University of Illinois in a fine talk on neck dissection during one of our programs. It is young men like these who will be the teachers of tomorrow and who will help to reshape and expand our surgical field. I cannot help but recall that our own former member, Dr. Joseph Beck, long a national figure in our field, always considered himself a surgeon of the head and neck. Were he here tonight, in person, as I hope he is in the spirit, I am sure that he would be the first one to approve a return to such a concept.

A few words should be said about another phase of the resident problem. The period of resident training including the basic course is now a minimum of three years. This time is none too long when it is considered that the future otolaryngologist will have to know both the old and the new in our field. It is very long if we remember that most of these men are 24 to 26 years of age at the time of graduation and that after finishing their internship most of them will have to spend a minimum of twenty-one months in the armed services. The effect of this is to discourage all except young men of well-to-do families from entering special fields. Without independent means, a resident cannot be married or even live outside of the hospital on the nominal salary he is paid. The net result is, in many cases, bad for the morale of the student and bad for the future of medicine, since it is precisely these young men who formerly assisted in our research. It is my conviction that residents should be paid a living wage like other full-time members of the teaching staff and that this expenditure should be considered a normal part of the budget of every teaching institution.

One hears nowadays that otolaryngology is a dying specialty. In fact, sometimes one hears worse than that. The other day I mentioned to a medical friend that I was going to talk on the state of otolaryngology, and he said, "Oh, you are going to read the obituary." He considered us not merely dying but dead and buried. I think that people who take this point of view place far too much emphasis on a few surgical procedures and too little on the history of our specialty. When I was in Vienna in 1927, there were lecturers who could still remember when it was customary to treat suppuration of the maxillary sinus through an opening made by pulling a tooth. How many different methods of treatment, how many different operations for this condition alone have been in vogue during the past sixty years! The truth is that our specialty, like most other medical fields, is in a state of constant ferment. There can and will probably be no permanence or stability in otolaryngology for many years, and we must be ready at all times to adapt ourselves to changing conditions.

For a long time we looked toward Europe for growth in our field. During the past twenty-five years this has been only partly true, and it is hard to believe that it will be true ever again. The exhaustion of two wars, the blight of Hitler on the Continent, and the paralysis of socialized medicine in England make it unlikely that any great progress, at least in the clinical phase of our field, will come from the Old World. It will be for American otolaryngologists and American teaching institutions to shape the future of our specialty. In this effort I am confident that the members of the Chicago Laryngological and Otological Society will play a worthy role.

Case Reports

BILATERAL ABDUCTOR PARALYSIS COMPLICATED BY FRACTURED RIBS

BERT DeBORD, M.D.
TEMPLE, TEXAS

BILATERAL abductor paralysis of the vocal cords is not encountered frequently and when associated with multiple rib fractures presents an interesting and unusual emergency.

On Aug. 11, 1950, N. V., a white woman aged 51, entered Scott and White Hospital in a semiconscious state with severe dyspnea, laryngeal stridor, and indrawing of the sternum. The husband revealed that the patient had had a thyroidectomy elsewhere in 1938 and was rather hoarse for three months following surgery. Since that time she had had dyspnea on exertion, and for the past two months the dyspnea had been unusually severe.

On examination paradoxical respiration was noted. Roentgenograms of the chest revealed a fracture of the left third, fourth, fifth, sixth, seventh, and eighth ribs in the posterior axillary line. Both vocal cords were fixed in the midline and approximated so as to obstruct seriously the air passage.

An immediate tracheotomy was performed. When the tracheotomy tube was inserted, breathing ceased. The tube was removed, and a bronchoscope was introduced as far as the carina and no obstruction was encountered except some spasm. Artificial respiration was begun, and after about two minutes breathing recurred. Oxygen was given, the bronchoscope was withdrawn, and the tracheotomy tube was reinserted. The patient reacted satisfactorily and had an uneventful convalescence. The ribs healed satisfactorily in about four weeks.

On September 25, an arytenoidectomy was performed on the right side in the following manner: A midline incision was made through the thyroid cartilage, and the two alae were retracted so that the interior of the larynx was well exposed. The right arytenoid cartilage was dissected free and removed. The mucosa was closed with 0000 absorbable surgical suture. The thyroid alae were then approximated, and the overlying muscles and skin were sutured. On October 24 the tracheotomy tube was removed and the tracheotomy stoma was closed.

Since the arytenoidectomy, the patient has gained 27 lb. (12.2 kg.), has had no dyspnea, even on exertion, and the voice is excellent.

The interesting occurrence of the multiple fractures of the ribs on the left side presumably was brought about by an intense, voluntary respiratory effort, as there was no history of previous trauma.

SUMMARY

A case of multiple rib fractures following bilateral abductor paralysis of 12 years' duration is presented. Complete rehabilitation ensued after a unilateral arytenoidectomy.

312 West Avenue G.

KING OPERATION FOR CEREBRAL ABSCESS

Report of a Case Complicating Chronic Mastoiditis, with Cholesteatoma and Labyrinthitis

WALTER D. CHASE, M.D.
BETHLEHEM, PA.

HOW MANY otologists who began practice before the advent of the recent fenestration operation and the most modern technique in dealing with intracranial complications of otitis media with chemotherapy and the antibiotics have had tragic results in dealing with brain abscess! During my training days, in Vienna and Budapest, while I studied with the, at that time, masters of otology, I strove to absorb the invaluable instruction those most-experienced teachers afforded me. After taking up practice, I very soon encountered cases requiring the greatest skill if gravely ill patients, suffering from the then common intracranial complications, were to be saved. Often I was thwarted in my most painstaking efforts. I can remember the little children who died from lateral-sinus thrombosis, meningitis, and brain abscess, in spite of my employment of the technique learned from my teachers.

Then I remembered the paper written by Dr. Joseph E. J. King [1] on his treatment of brain abscess. Before that, my complete adherence to the Vienna teachings had prevented me from adopting any but their techniques, but my continuing failures made me take stock, seeking ways for improvement. Dr. King showed me his slides, using diagrams to illustrate his points, and a volume on brain abscess by the revered Macewen [2] and then allowed me to visit his patients with him.

Dr. King demonstrated the fact that when a drain is inserted into an abscess in the jelly-like brain substance, parts of the wall of the abscess tend to collapse, making clover-leaf-like spaces which are incompletely drained. Even when encapsulated, a drain allows the walls to sag and there are pockets which tend not to remain in contact with the drained area, thus making for artificial locular division of the abscess cavity with later so-called relapse or recurrence. On the other hand, when the abscess is uncapped the natural tendency for herniation eliminates the abscess cavity and constitutes self-drainage, which Dr. King practiced. Later, he adopted the Mikulicz drain, which is the ideal method of treatment, unless the more recent enucleation technique is employed when encapsulation favors this method.

When I read of surgeons employing a simple drainage tube or aspirating the abscess contents and then removing the cannula, perhaps to repeat the process later, I cannot but believe the successful outcome represents an exceptionally lucky occurrence and that the technique, if employed regularly, would bring failures in the majority of cases. Complete drainage by the King technique or enucleation of the encapsulated abscess is necessary if the greatest number of cures is to be expected, I feel confident. Another point must be admitted: Patients handled by

1. King, J. E. J.: The Treatment of Brain Abscess: A Surgical Technic in Which the Usual Drainage Methods Are Avoided, Arch. Otolaryng. **1**:26 (Jan.) 1925.

2. Macewen, W.: Pyogenic Infective Diseases of the Brain and Spinal Cord, Glasgow, J. Maclehose & Sons, 1893.

an inferior technique are cured nowadays by the inestimable influence of the powerful antibiotics and chemotherapy, plus transfusions and the many other intravenous medications at present available and customarily used, which makes for the impression that the incomplete drainage methods formerly (and still occasionally) employed are admissible.

The Török fenestrated glass drain was also successfully employed by Dr. Török in many cerebellar abscesses, one such case having been reported by me in 1924.[3] As Dr. King described them, cerebellar abscesses are usually anchored to the petrous bone, when of otological origin, and the Mikulicz drain is not employable in these cases.

When Dr. King reported his unprecedented successes he was without the services of penicillin and the sulfonamides and he relied upon keeping the field bathed in diluted sodium hypochlorite solution (modified Dakin's solution®). Thus limited, his results were phenomenal.

The King method in cases of temporosphenoidal abscess was to make an opening through the skull beyond the mastoidectomy field by an incision joining a point 1½ in. above the external meatus to a point 1 in. behind the external meatus, expose the skull, trephine through the bone, nick the dura, insert an exploring instrument through the cortex, and feel for the capsule of the abscess. He described the feeling on contacting the abscess as "like touching a soft rubber ball." The presence of the abscess thus verified, the bony opening was made "about the size of a silver dollar," the dura excised to a size about 1 cm. less around the border, and then the brain substance overlying the abscess removed by suction. The top of the capsule was removed, and the contents of the abscess were removed by sponging or suction. Then a Mikulicz drain of iodoform gauze was inserted, the contents of this "handkerchief" filled with strips of this gauze, and so on, as described in his paper.[4] At that time the dressings overlying the Mikulicz drain were kept wet with diluted sodium hypochlorite solution. The desirable herniation which followed, insuring complete drainage, was prevented from becoming excessive by spinal taps and enemas and saline cathartics. Dr. King had phenomenal success, even saving patients in whom the abscess had ruptured into the ventricle.[5]

My next patient with brain abscess came to me on May 15, 1947—a 25-yr.-old man with chronic mastoiditis which had become complicated with labyrinthitis five weeks before. Four days before coming to me he vomited four times and had vertigo and pain over the right (involved) side of the head and mastoid. He walked into my office supported by his wife. Examination showed foul discharge from the right ear; external canal partly filled with granulations; tenderness but no swelling over the mastoid, and a swaying, independent of the position of the head. He past-pointed left with his left hand only; there was left nystagmus of brief, horizontal-rotatory type after testing for left "fixation" nystagmus; the pupils were equal and reacted to light and in accommodation. Since he became nauseated, no caloric test was made and he was immediately hospitalized. At 1 p. m. his temperature was 99 F., pulse rate 76, and respiration rate 23. Sulfathiazole and sufadiazine, 0.5 gm. each, were given every four hours. The urine was normal. Blood examination showed 7,700 white blood cells, 4,900,000 red blood cells, and hemoglobin, 13 gm. On May 18, his temperature was 98 F. at the lowest and the pulse rate 60 for the next four days. The pulse rate then reached 80 to 90 for three days, fell

3. Chase, W. D.: Cerebellar Abscess: Report of an Interesting Case, with Description of Therapy as Practiced by Prof. Török of Budapest, Atlantic M. J. **28**:742 (Aug.) 1925.

4. King, J. E. J.: Brain Abscess, Ann. Surg. **103**:647 (May) 1936.

5. King, J. E. J.: Brain Abscess with Gross Rupture into the Lateral Ventricle, South. Surg. **8**:521 (Dec.) 1938.

again to 60 until May 28, when it rose to 70 to 80, and rose to 98 on May 29, on which date his temperature was 97 F.

A radical mastoid operation was done May 26. Sclerotic type was found, but the first penetration of the mastoid gouge encountered pus under pressure, there being a large epidural abscess of the middle fossa, with the roof of the tympanum, antrum, and the rest of the mastoid cavity having been destroyed. The dura was covered with thick, shaggy granulations. At the site of the horizontal semicircular canal there was a deep fissure and the posterior labyrinth area looked eroded and had some black specks. Granulations filled the tympanum, and a large cholesteatoma occupied the enlarged antral space. The lateral sinus lay far forward and superficially, but it had not become involved.

The floor of the middle fossa was removed beyond the perforation until healthy bone appeared, the posterior canal wall was taken down in the pattern of radical mastoidectomy, the middle-ear granulations were scrubbed away with iodoform gauze, sulfonamide powder was sprinkled into the wound, and iodoform gauze was packed loosely into the operative field. Three loose skin sutures were taken, the wound being left "wide open," and external dressings were applied.

On May 29, the patient's temperature dropped to 97 F., and the pulse rate was 98. The temperature remained normal thereafter, and the pulse rate was 60 to 80 until June 1 when it became 98, dropped to 80, then rose to 90. On June 2, plastic closure of the mastoid wound was done. The patient went home June 7.

On June 16, he returned with a severe headache, nausea and vomiting, dizziness, and pain "in the eyes." His temperature was 98 F., the pulse rate 98, and the respiration rate 22. A brain abscess was evidently present, as had been expected. On June 20, a cold caloric test of the right labyrinth gave a negative result, the vestibular portion of that internal ear having been destroyed by the infection. The location of the abscess was the main problem in the diagnosis, determination of whether it was in the posterior or the middle fossa. The preceding labyrinthitis directed attention to the posterior fossa, while the finding of damage to the dura of the middle fossa at the mastoid operation directed attention to the middle fossa. Dr. A. B. Hamilton, neurologist, favored the latter, believing the abscess to be in the temporal lobe. The ophthalmologist, Henry K. Erwin, found a choked disk of 2 degrees on the right and 1 degree on the left.

The patient was given penicillin and watched for a favorable time (encapsulation evidence) for exploration and drainage. On June 23, exploration, under local anesthesia, was made through an incision starting 1½ in. above the external meatus and carried backward and downward to a point 1 in. behind the external meatus. The squamous portion was removed with a rongeur from the previously made middle-fossa exposure upward for the necessary distance. Then the dura was opened and a probe (a small, slightly curved Eustachian catheter) introduced through the cortex. At the depth of about 2 cm. the abscess was encountered, the feeling of "a soft rubber ball" being experienced definitely, as Dr. King had described it.

The dura as well as the bony opening were then removed over the abscess to the prescribed degree, and the overlying brain substance was removed with a tonsil-suction instrument. The capsule also appeared as Dr. King had described it—"dark-red, like a slightly over-ripe cherry." Then the top of the capsule was grasped with the forceps and removed with a diathermy cautery, the contents of the abscess, which was the size of a hen's egg, were removed by suction, and the inside of the capsule was gently sponged. The Mikulicz dressing was applied and sterile gauze bound on. The patient's condition was very good.

The subsequent herniation was not so easily controlled as usual, as Dr. King described, and large amounts of spinal fluid had to be removed at frequent intervals, at one time the Love indwelling spinal catheter being used for a short time. With this treatment the patient recovered.

The closure of the scalp wound presented a problem but was accomplished by encircling the site by an incision through the scalp and dissecting toward the center, thus avoiding the chance of penetrating the dura. After sufficient undermining of the scalp, it was readily brought together and closed with nonabsorbable surgical suture.

The patient regained his health completely and resumed his occupation as a printer. An audiometer test of the right ear March 25, 1948, showed about 40% loss of hearing in frequencies 64 to 1020, 60% at 2896, 25% at 8192, and 45% at 11584.

230 E. Broad St.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1950

PERORAL ENDOSCOPY

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ANATOMIC and physiologic studies of the tracheobronchial tree and esophagus are necessary so that information gained may later be applied clinically. By these observations the clinician is guided in his search for the correct diagnosis and proper treatment, and new thoughts and interest in new fields of endeavor are stimulated.

PHYSIOLOGY OF TRACHEOBRONCHIAL TREE

Any addition to the knowledge of the underlying physiology of respiratory obstruction is a basic contribution to the understanding of this problem. Gray [1] demonstrated that the first change resulting from respiratory obstruction was a reduction in the ventilation capacity of the pulmonary bellows, which if mild meant dyspnea on exertion and if severe meant dyspnea even at rest. The second respiratory change was hypoventilation, resulting in an increased oxygen consumption being necessary to support the extra respiratory effort. As hypoventilation became severer, it caused first a retention of carbon dioxide, second a fall in the oxygen saturation of the arterial blood, and third an acidemia, or fall in the blood pH. If the obstruction and resulting hypoventilation continued to progress, a crisis was reached where the asphyxial blood changes ceased to behave as respiratory stimuli, and the breathing effort was inhibited. Oxygen therapy is important in the treatment of respiratory obstruction, but it cannot correct all the asphyxial changes. Oxygen therapy cannot correct the acidosis or prevent the development of pulmonary complications which arise secondary to obstruction. This can be accomplished only by relief of the respiratory obstruction and maintenance of a patent airway.

Hilding [2] was of the opinion that air could be completely absorbed from almost any space or tissue in the body, such as obstructed portions of lung, provided sufficient time is allowed and provided the space containing the air can collapse. If the space containing the air cannot collapse or can do so only partially, a negative pressure develops, depending upon the gas pressures present in the absorbing fluid. In postoperative atelectasis there are at least three mechanisms by which the air

1. Gray, J. S.: The Physiology of Respiratory Obstruction, Ann. Otol. Rhin. & Laryng. **59**:72 (March) 1950.

2. Hilding, A. C.: The Removal of Air from the Respiratory Tract and Certain Other Body Spaces Under Normal and Abnormal Conditions, Ann. Otol. Rhin. & Laryng. **59**:309 (June) 1950.

can be removed: (1) absorption, (2) displacement by secretion, and (3) the pumping action of the cilia and moving masses of mucus. The negative pressure in most cases of postoperative atelectasis is, in all probability, maintained solely by ciliary action.

Although the negative pressures obtained in the experiments performed by Ballenger [3] were less than those obtained by Hilding, his studies confirmed the observation that the respiratory cilia can, under special circumstances, develop a negative pressure by moving a mass of mucus.

Electrical stimulation of the tracheobronchial mucosa, accomplished during bronchoscopy on 25 patients by Morton and associates,[4] indicated that the linings of the trachea and bronchi were sensitive and that pain resulted from stimulation. The areas in which pain of tracheobronchial origin is felt was charted. In general, pain arising from the bronchial tree was referred to the homolateral anterior chest within 2 to 4 cm. of the sternum or to the anterior cervical region within 2 cm. of the midline. Pain of tracheal origin was felt in the midline anteriorly at sites extending from the larynx to the xiphoid process.

ANATOMY

A terminology common to the internist, thoracic surgeon, and bronchoesophagologist would simplify problems of diagnosis and treatment of chest conditions. Neil [5] advocated universal adoption of the terminology of bronchopulmonary segments approved at the Pan-Pacific Surgical Congress in 1948: upper lobe: apical, lateral, anterior, subapical; middle lobe: anterior, lateral; lower lobe: apical, subapical, cardiac, anterior basal, lateral basal, posterior basal.

BRONCHOSPIROGRAPHY

Bronchospirography concerns the bronchologist because of his interest in bronchopulmonary physiology and pathology. He also will see patients for whom functional tests are indicated and will probably participate in the procedures. Leiner and Liebler [6] found bronchospirometric studies valuable in obtaining the following data: oxygen intake, minute volume, ventilation equivalent, tidal air, vital capacity, complemental air, and reserve air. These studies are indicated when irreversible operations on one lung are planned and any doubt exists as to the function of the other lung.

Long and her co-workers [7] studied by bronchospirometry the ventilation and oxygen uptake in a group of 64 patients with bronchiectasis and five patients with other pulmonary disease; these studies were compared with those of 63 normal subjects. It was found that the maximum breathing capacity was reduced in bronchiectasis below normal in a manner roughly proportional to the extent of the

3. Ballenger, J. J.: The Producing of Negative Pressures by Respiratory Cilia, Ann. Otol. Rhin. & Laryng. **59**:694 (Sept.) 1950.

4. Morton, D. R.; Klassen, K. P., and Curtis, G. M.: The Clinical Physiology of the Human Bronchi, Surgery **28**:699 (Oct.) 1950.

5. Neil, J. H.: Azygos Lobes of the Lung, and Terminology of the Bronchopulmonary Segments of the Bronchial Tree, Ann. Otol. Rhin. & Laryng. **59**:409 (June) 1950.

6. Leiner, G. C., and Liebler, J. B.: The Technique of Bronchospirometry, Dis. Chest **17**:578 (May) 1950.

7. Long, J. H.; Norris, C. M.; Burnett, W. E., and Wester, M. R.: Bronchospirometric Studies in Bronchiectasis, Before and After Lobectomy, J. Thoracic Surg. **19**:477 (March) 1950.

disease. The over-all ventilation was normal only during quiet respiration and was reduced during effort. Postoperatively further loss of function on the diseased side occurred in the majority of patients but was balanced by increasing functions in the opposite, undiseased lung.

ANESTHESIA

Although general, local, and intravenous anesthesia have been used in peroral endoscopic procedures, topical anesthesia has been more generally used, because of its relative safety and the ability to secure patient cooperation. However, in suspension laryngoscopy topical anesthesia rarely suffices, and it is necessary to employ general or supplemental intravenous anesthesia to secure satisfactory working conditions. Adequate anesthesia for suspension laryngoscopy requires almost complete depression of laryngeal activity and profound muscular relaxation. A method of anesthetization employing a combination of cocaine, thiopental (pentothal®) sodium, and curare was described by Davis.[8] Preliminary topical cocainization to reduce sensory stimulation was secured by the application of 10% cocaine to the pyriform fossae and vocal cords. The intravenous use of thiopental sodium provided additional sensory depression, and the associated use of curare intravenously aided muscular relaxation and helped prevent laryngeal spasm and coughing. Reflex laryngeal spasm from tracheal stimulation was not prevented by this method but was minimized by careful aspiration and prevention of the entrance of blood and secretions into the trachea. Since the method possesses the potential dangers of persistent laryngeal spasm and overdosage of thiopental sodium or curare, given in a vain attempt to provide exposure, it should be employed only by those who are familiar with the drugs used and have adequate resuscitative measures available.

General anesthesia for bronchoscopic examinations was reported by Clark and Goodwin.[9] The recommended procedure consisted in premedication with pantopon® (total alkaloids of opium as soluble hydrochloride) and scopolamine hydrobromide, followed in one to one-half hours with thiopental sodium ("thiopentone") given intravenously and continuous oxygen therapy as soon as there was loss of consciousness. The larynx was then anesthetized with a 10% cocaine spray, and an intratracheal tube of the Magill type was inserted, with subsequent spraying of cocaine into the tracheobronchial tree, after which bronchoscopy was performed.

TRACHEOBRONCHIAL ASPIRATION

Most cases of acute respiratory distress in newborn infants fall into three groups: (1) those in which the condition is of central nervous system origin; (2) those in which it is obstructive in nature, and (3) those in which anomalies are found. Emerson[10] advocated bronchoscopy for the third group, which is small in number, in addition to the second, and largest, group, for which aspiration of the tracheobronchial tree is essential. He developed a relatively simple technique which can be used by the obstetrician or anesthetist, and upon a checking of the efficiency

8. Davis, D. A.: The Induction of Anesthesia for Suspension Laryngoscopy with Cocaine, Sodium Pentothal and Curare, Anesthesiology **11**:245 (March) 1950.

9. Clark, J. V., and Goodwin, G. P.: Anesthesia and Surgical Procedure for Bronchoscopy, J. Laryng. & Otol. **64**:570 (Sept.) 1950.

10. Emerson, E. B., Jr.: A Safe and Positive Method of Tracheobronchial Aspiration in the Newborn, J. A. M. A. **144**:613 (Oct. 21) 1950.

of this method, in many cases by immediate bronchoscopy, the tracheobronchial tree was found to be clean and unobstructed. In this technique a No. 11 F. ureteral catheter with a cylindrical tip, multiple side openings, and an oblique open end was passed between the vocal cords under direct vision after exposure of the larynx with a direct laryngoscope. Continuous suction was maintained for a short period and continued as the catheter was withdrawn. When used outside of the hospital a small rubber bulb may be used to furnish suction. (This method provides a quick, efficient, and safe method of tracheobronchial aspiration.)

Samson and Dugan [11] emphasized the need for continuous tracheobronchial patency using aspiration when necessary and outlined techniques and indications for this procedure in both infants and adults. Although bronchoscopy with aspiration and mucosal shrinkage is the most effective means of achieving this end, under a variety of conditions complementary and supplementary procedures are necessary if the patient can not clear his own airway by coughing. The indications for primary bronchoscopy in adults were listed for certain conditions: (1) for inspissated secretions; (2) for blood clots; (3) for suspected particulate matter, as in the aspiration of stomach contents; (4) for tracheal obstruction, and (5) for pulmonary atelectasis which is lobar or greater in extent. A patient who is completely exhausted, without the strength to cough, and whose airway is filled with secretions needs bronchoscopy, rather than catheter aspiration. Because of the poorly developed cough mechanism, in infants the prime indication for bronchoscopy is excess secretion. The technique employed was flooding of the hypopharynx with oxygen after exposure of the larynx with a direct laryngoscope. An aspirating tube with thumb control was then passed, without suction, into the bronchial tree. As the aspirator was withdrawn into the trachea, intermittent suction was applied. To enter the left bronchus, the head was turned to the right and the lip of the laryngoscope to the left.

Buyers [12] described the use of a double-lumen intratracheal tube for the simultaneous administration of oxygen deep in the tracheobronchial tree and the aspiration of secretions. The tube is not only useful in intermittent aspiration but may be left in the trachea as long as secretions must be removed and concomitant oxygen therapy administered.

Bronchoscopy in the newborn has become an accepted endoscopic procedure and has proved particularly valuable in atelectasis, which is prone to occur in the premature infant. Conservative management was first employed, and when the condition failed to respond, bronchoscopy was performed by Doust [13] in six cases.

In discussing the assistance which the endoscopist can given the obstetrician and pediatrician in the handling of problems of resuscitation and aspiration of the newborn, Blaisdell [14] emphasized the need for familiarity with pediatric practice in the management of such problems and the necessity for the endoscopist to be available for these emergencies. The endoscopist who is conversant with other disorders

11. Samson, P. C., and Dugan, D. J.: Tracheobronchial Toilet in Infant and Adult, Ann. Otol. Rhin. & Laryng. **59**:738 (Sept.) 1950.

12. Buyers, R. A.: A Double Lumen Intratracheal Tube for Simultaneous Oxygen Administration and Tracheobronchial Aspiration, Surgery **27**:74 (Jan.) 1950.

13. Doust, A. W.: Bronchoscopy in Atelectasis in the Newborn, Laryngoscope **60**:207 (March) 1950.

14. Blaisdell, I. H.: The Endoscopist and the Newborn Infant, Ann. Otol. Rhin. & Laryng. **59**:907 (Dec.) 1950.

of the newborn that simulate respiratory obstruction is better able to make an accurate diagnosis and institute proper treatment, for in each individual case it must be decided whether or not endoscopy is indicated. In these delicate infants all manipulations must be gentle; the infants must be kept warm during all procedures, and it is unwise to transport them to distant operating and roentgen rooms.

Unusual cases in which pulmonary atelectasis was the initial diagnosis were discussed by Priest.[15] On further study the diagnosis proved to be some other condition: idiopathic pulmonary hypoplasia, pulmonary hypoplasia associated with congenital heart disease, pulmonary hypoplasia accompanying diaphragmatic hernia, unilateral pulmonary agenesis, traumatic bronchial rupture, and atelectasis due to bronchial asthma. Bronchoscopy was helpful in the diagnosis in all the cases, and bronchography was an aid in some.

TRAUMA OF TRACHEA

In reporting a case of rupture of the trachea occurring in a child during bronchoscopy, Andrews and McMahon[16] emphasized the responsibility of the endoscopist in positioning of the patient and directing the head and shoulder holder. Failure of rotation of the head due to uncontrollable movements of the child resulting in vertical movements of the head was the principal cause of the rupture. In spite of the shoulder holder, the patient moved his shoulders up and down upon introduction of the bronchoscope. After the instrument was in the trachea, a web across the right posterolateral area of the field with a small amount of blood was seen, and the procedure was immediately terminated. Within three minutes subcutaneous emphysema appeared and rapidly progressed over the entire body, head, and extremities. The movement of the head and shoulders while the bronchoscope was in the trachea brought about an angle with the horizontal, and the small size of the instrument applied this strain over a small area, resulting in rupture between the tracheal rings. The factors influencing the successful treatment of this complication were oxygen therapy, bilateral chest aspiration, and mediastinotomy.

FOREIGN BODIES IN TRACHEA

An unusually large tracheal foreign body (fountain-pen cap) was reported by Dickey.[17] There were no immediate signs of respiratory obstruction, and these developed suddenly several hours later. The author recommended removal of large foreign bodies through a tracheotomy opening rather than subjection of the larynx to the trauma incident to peroral removal.

TRACHEAL STENOSIS

A series of 29 cases of benign tracheal stenosis due solely to lesions of the tracheal wall was reported by Holinger and his associates.[18] Twelve were congenital

15. Priest, R. E.: Unusual Conditions Simulating Pulmonary Atelectasis, Ann. Otol. Rhin. & Laryng. **59**:889 (Dec.) 1950.

16. Andrews, A. H., Jr., and McMahon, R. J.: Rupture of the Trachea During Bronchoscopy and Principles of Positioning for Endoscopic Procedures, Ann. Otol. Rhin. & Laryng. **59**:726 (Sept.) 1950.

17. Dickey, L. C.: An Unusually Large Tracheal Foreign Body, Laryngoscope **60**:392 (April) 1950.

18. Holinger, P. H.; Johnston, K. C., and Basinger, C. E.: Benign Stenosis of the Trachea, Ann. Otol. Rhin. & Laryng. **59**:837 (Sept.) 1950.

in origin, and 17 were due to acquired lesions. The congenital anomalies producing stenosis were divided into the congenital fibrous strictures and those due to absence or deformity of the tracheal cartilages. Webs, fibrous strictures of segments of the trachea, and stenoses associated with congenital esophageal atresia and tracheoesophageal fistula were included in the first group. Tracheomalacia, cartilaginous deformities, and persisting deformity stenoses associated with vascular rings were included in the second group. Of the acquired tracheal stenoses, six were due to nonspecific inflammatory processes. The specific inflammatory diseases considered as etiologic factors in tracheal stenosis were syphilis, tuberculosis, diphtheria, typhoid, and rhinoscleroma. Five cases of stricture of the trachea of traumatic origin were presented. In two, fractures of the thoracic portion of the trachea occurred in automobile accidents. Two were postsurgical strictures of the cervical trachea, and one was a golf-ball fracture of the cervical trachea. The diagnosis of benign tracheal stenosis was suspected from the history and physical findings. Roentgen and endoscopic examinations were essential in establishing the diagnosis.

TUMORS OF TRACHEA

In reporting a case of primary carcinoma of the trachea, Guttman and co-workers [19] pointed out that this condition is rarely considered as a possibility when the symptoms of increasing dyspnea and wheezing are present but is usually misdiagnosed as asthma. In this case staining by the method of Papanicolaou of secretions of both the sputum and the exudate, obtained by direct suction of the lesion itself through the bronchoscope, gave negative results, the diagnosis being made by examination of biopsy material.

Twenty-eight cases of tracheal tumors were reported by Holinger and his co-workers.[20] Eighteen of the tumors were primary in the trachea, and 10 invaded the trachea from adjacent structures to give signs, symptoms, and endoscopic findings of primary tracheal tumors. Five of the tumors were inflammatory, and 23 neoplastic, of which 5 were benign and 18 malignant. The symptomatology was similar in most cases. The first symptom was generally a dry, hacking cough, followed by wheezing which was frequently expiratory and bilateral in character. Hemoptysis was a constant finding in the patients with malignant tracheal tumors but was only occasionally present in patients with benign tumors. As the lesions progressed, dyspnea and orthopnea increased. Hoarseness was present in the later stages of the malignant tumors because of extension to involve the recurrent laryngeal nerves. Dysphagia was a rare accompanying symptom. Diagnostic studies included posteroanterior and lateral roentgenograms of the neck and chest, fluoroscopic examinations, and planograms. The final diagnosis was made by tracheoscopic examination and the histologic examination of tissue removed. The primary treatment of most patients was establishment of the airway. This was done by forceps, tracheotomy, snare, or coring through the tumor with the bronchoscope. Subsequent endoscopic treatment consisted in electroresection and coagulation, radon-seed

19. Guttman, M. R.; Simon, M. U., and Nitka, C. B.: Primary Carcinoma of the Trachea, Arch. Otolaryng. **52**:172 (Aug.) 1950.

20. Holinger, P. H.; Novak, F. J., and Johnston, K. C.: Tumors of the Trachea, Laryngoscope **60**:1086 (Nov.) 1950.

implantation, and repeated forceps removal. External irradiation was employed in the cases of malignant tumor, and cervical tracheal resection was successfully used in one case.

TRACHEOTOMY

In addition to its place in the relief of dyspnea from laryngeal obstruction, tracheotomy is now finding other uses, namely, to afford a means for tracheobronchial aspiration in postoperative patients and in those who, in certain conditions, are unable to get rid of secretions.

Echols and co-workers [21] advocated tracheotomy in the management of severe head injuries and considered this procedure superior to any other method of maintaining effective aeration of the lungs in unconscious patients. It was advised for every patient unconscious from a head injury if it seemed likely that the coma would persist more than 24 hr. and if nonsurgical methods of maintaining an adequate airway were not effective. Of the 108 tracheotomies performed during a three-and-one-half-year period, only 39 were performed because of obstructive lesions.

Reynolds and associates [22] reported 11 cases of esophagectomy, in six of which tracheotomies were performed postoperatively for aspiration purposes. This procedure was considered to be greatly beneficial, and it was recommended postoperatively in all cases after excision of the upper and middle portions of the esophagus when there was retention of mucus in the tracheobronchial tree which could not be readily handled by the more conventional means of transoral suction.

Because many fatal complications of tetanus are respiratory in origin, Creech and his associates [23] recommended early tracheotomy to overcome the respiratory difficulty. Formerly, profound sedation was used to control the muscle irritability in tetanus, but the sedation itself predisposed to respiratory complications. The authors feel that deep sedation is not necessary if an open airway is maintained by tracheotomy.

BRONCHIAL OBSTRUCTION

Jackson [24] presented a review and formulation of the bronchoscopic phases of bronchial obstruction. Bronchial obstruction acts as a primary, a predisposing, and a perpetuating factor in the production of bronchial and pulmonary disease. The mechanism by which normal rhythmic respiratory movements produced valvular types of obstruction that caused emphysema and atelectasis in the tributary areas has been termed the "expansile check-valve." The expansile check-valve mechanism is seen in practically every disease of the lung and occurs not only in endobronchial conditions but in compression stenoses as well. Frequent causes of bronchial obstruction are pathologic secretions and exudates that the cough reflex cannot expel because of their increased adhesiveness. Difficulty of expulsion is increased by accumulation which occurs when the cough reflex is inefficient from lack of glottic cooperation, or suppressed by toxemia, alcohol, or drugs, especially opiates and

21. Echols, D. H.; Llewellyn, R.; Kirgis, H. D.; Rehfeldt, F. C., and Garcia-Bengochea, F.: Tracheotomy in the Management of Severe Head Injuries, Surgery **28**:801 (Nov.) 1950.

22. Reynolds, J. T.; Holinger, P. H.; Andrews, A. H., Jr.; Young, J. P., Jr., and Marlowe, W. H.: Role of Tracheotomy in the Postoperative Care of Patients Subjected to Esophagectomy, Arch. Surg. **61**:211 (Aug.) 1950.

23. Creech, O.; Woodhall, J. P., and Ochsner, A.: The Necessity for Tracheotomy in the Treatment of Tetanus to Prevent Lethal Respiratory Complications, Surgery **27**:62 (Jan.) 1950.

24. Jackson, C.: Bronchial Obstruction, Dis. Chest **17**:125 (Feb.) 1950.

other sedatives. Opiates and atropine promote bronchial obstruction in four different ways: (1) the desiccating effect of these drugs greatly increases the adhesiveness of pathologic secretions and exudates; (2) the desiccation favors coagulation into firm plugs; (3) by suppression of the cough reflexes, there is accumulation, coagulation, and plug formation, and (4) by suppressing cough, the natural defense machinery of the lungs is crippled. Aspiration of the tracheobronchial tree can be done with a silk-woven aspirating tube inserted through the laryngoscope or with a bronchoscope. Deep insertion of the aspirating tube is unnecessary, for the tussive squeeze will force exudates up into the large bronchi, from which they can be aspirated. Prompt arrest of beginning stagnation of pathologic secretions after acute respiratory infections will prevent chronic bronchitis and bronchiectasis, which occur so often as sequelae of acute infections. In all forms of bronchial obstruction the relations of the bronchoscope to treatment are those of a speculum through which treatment may or may not be indicated. As a means of treatment its successful removal of foreign bodies gives bronchoscopy a unique position. Two important duties of the bronchoscopist in all kinds of cases are to maintain constantly a clear and adequate airway to the alveoli and to reestablish normal physiologic peroral drainage.

ACUTE LARYNGOTRACHEOBRONCHITIS

There is a lack of full agreement regarding the best methods of treatment of acute laryngotracheobronchitis, which remains a serious disease. The use of sodium lauryl sulfate (duponol C®) as a wetting agent to combat crusting in the trachea and bronchi was recommended by Gorman.[25] Mention was made of several patients with laryngotracheobronchitis that were considered to be in need of immediate tracheotomy but recovered without tracheotomy after the introduction of sodium lauryl sulfate in the water of the vaporizer.

PULMONARY HODGKIN'S DISEASE

In a case report Heatly [26] called attention to the frequency of bronchopulmonary involvement in Hodgkin's disease and its simulation of other pulmonary conditions, notably adenoma and carcinoma. Pulmonary involvement is frequent in Hodgkin's disease, and in typical cases the associated enlargement of superficial lymph nodes permits accurate diagnosis by biopsy. Uncertainty may arise in cases in which external lymphomas are poorly developed or altogether absent. The lesions of Hodgkin's disease both clinically and roentgenologically may closely simulate other pulmonary conditions. A significant degree of bronchial involvement must be considered among the characteristic features of the pulmonary manifestations of the disease. This may take the form of external compression of the bronchus or infiltration and invasion producing endobronchial lesions. Occasionally, as in the case reported, even the histologic studies of biopsy material may not be conclusive. The response of Hodgkin's granulomas to roentgen therapy may furnish valuable evidence in the differential diagnosis of these more obscure cases.

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25. Gorman, J. R.: The Use of a Wetting Agent in Laryngotracheobronchitis with Report of Cases to Date, Laryngoscope **60**:510 (May) 1950.

26. Heatly, C. A.: Localized Pulmonary Hodgkin's Disease: Bronchoscopic Aspects, Ann. Otol. Rhin. & Laryng. **59**:705 (Sept.) 1950.

A case of obstructing intrabronchial Hodgkin's disease diagnosed by bronchoscopic biopsy was reported by Higginson and Grismer.[27] Whereas the microscopic diagnosis of the tissue obtained at the first bronchoscopy was inflammation, a second specimen was taken, and the true diagnosis was established.

TRACHEOBRONCHIAL TUBERCULOSIS

A group of 49 patients with active endobronchial tuberculosis who were treated with streptomycin and followed to healing were reported by Erman.[28] The majority of the patients received 1 gm. of streptomycin intramuscularly daily. The average healing time for hyperplastic endobronchial tuberculosis was 42 days, for submucosal endobronchial tuberculosis 42 days, and for ulcerative endobronchial tuberculosis slightly longer. All patients recovered without residual fibrosis except three patients with ulcerative tuberculosis, and these recovered with some degree of bronchial stenosis.

BRONCHOLITHIASIS

In a report of 41 cases of broncholithiasis by Schmidt and his associates,[29] the diagnosis was made in 15 of the cases on the basis of a history that the patient had coughed up a broncholith; in 18 cases the diagnosis was made at bronchoscopy, and in the other eight it was made at operation. The symptoms in the order of frequency were (1) cough, (2) hemoptysis, and (3) thoracic pain. On the roentgenogram the diagnosis of broncholithiasis was suspected in only 3 of the 41 cases. In retrospect, the most reliable diagnostic roentgenologic finding, when it occurred, was a dense area of calcium deposit at the apex of a triangular portion of collapsed lung. Of the 35 patients who underwent bronchoscopy, 32 had positive findings, consisting of a mass of granulation tissue overlying the stone, bronchial stricture, or palpation or visualization of the broncholith. Since broncholiths occur in association with carcinoma, tissue biopsy is essential, and operation was performed in eight cases of this series because bronchogenic carcinoma could not be ruled out prior to operation. In 17 instances the broncholith was either found and removed at bronchoscopy or coughed up immediately after bronchoscopy; in one case a broncholith was found but could not be removed bronchoscopically.

LUNG ABSCESS

An unusual case of lung abscess secondary to a hiatal hernia was reported by Coodley.[30]

FOREIGN BODIES IN THE BRONCHUS

A series of 160 cases of foreign bodies in the tracheobronchial tree was analyzed by Baylin and Martin.[31] Peanuts, seeds, and raw foods were the most frequent

27. Higginson, J. F., and Grismer, J. T.: Obstructing Intrabronchial Hodgkin's Disease, J. Thoracic Surg. **20**:961 (Dec.) 1950.

28. Erman, E. D.: Treatment of Endobronchial Tuberculosis with Streptomycin, J. Thoracic Surg. **20**:51 (July) 1950.

29. Schmidt, H. W.; Clagett, O. T., and McDonald, J. R.: Broncholithiasis, J. Thoracic Surg. **19**:226 (Feb.) 1950.

30. Coodley, E. L.: Pulmonary Abscess as Complication of Hiatal Hernia, Dis. Chest **17**:102 (Jan.) 1950.

31. Baylin, G. J., and Martin, T. L.: The Clinical and Roentgen Aspects of Non-Opaque Pulmonary Foreign Bodies, North Carolina M. J. **11**:59 (Feb.) 1950.

foreign objects, and there was close correlation between the physical and the roentgen findings. The mortality rate in this series was 5.6%.

A patient with unsuspected foreign body (paper clip) in the bronchus who had been treated for bronchial asthma for four years was reported by Bannon.[32] Roentgenograms of the chest, taken for suspected pneumonia, revealed the foreign body.

A case of unsuspected foreign body (grass head) in the bronchus which was not detected by repeated roentgen studies, bronchoscopies, bronchograms, or planograms was reported by Myerson.[33] The diagnosis was made after surgical exploration, when the foreign body was found in the excised pulmonary lobe.

In a review of the subject of magnet extraction of foreign bodies, Whalen[34] stated the opinion that this method provided a simple and effective means of dealing with magnetizable foreign bodies. The improvement in the Alnico permanent magnet, the biplane fluoroscope, increased knowledge of bronchopulmonary segmental anatomy, and intestinal intubation have all contributed help in the endoscopist's problem of removal of ferromagnetic foreign bodies from the air and food passages. The armamentarium of the endoscopist has increased to include an Alnico magnet that can be attached to an intestinal tube, a vertebrated-tip magnet for use in the smaller divisions of the bronchial tree, and a Penta type electromagnet for the removal of ferromagnetic foreign bodies from the stomach.

BRONCHIAL ADENOMA

Bronchial adenomatous lesions still remain problems in diagnosis and treatment. After studying 86 cases of bronchial adenoma, Moersch and McDonald[35] considered this lesion to be a low-grade carcinoma that possesses the ability to metastasize. In this series there were five cases with proved metastasis and three others with probable metastasis. The great majority of adenomas occurred in the larger bronchi and were accessible to bronchoscopic study. The type of treatment depended upon the situation of the lesion, the degree of involvement of the bronchial wall, the age of the patient, and the degree of secondary suppuration. A pedunculated adenoma accessible to bronchoscopic manipulation was treated by that method. Patients of advanced years and those whose lesion was located near the carina, so that a pneumonectomy would have to be performed, were also treated bronchoscopically. Surgical treatment was used in all other cases and in cases in which there was recurrence. After bronchoscopic treatment repeated bronchoscopic examinations were made to detect any recurrences.

UNUSUAL PULMONARY TUMORS

A case of leiomyoma of the lung with the single symptom of cough was reported by Williams and Daniel.[36] The roentgenogram revealed a dense opacity lying

32. Bannon, F. M.: Unsuspected Foreign Body of Bronchus, Connecticut M. J. **14**:696 (Aug.) 1950.

33. Myerson, M. C.: Unusual Foreign Body in a Secondary Bronchus, Ann. Otol. Rhin. & Laryng. **59**:760 (Sept.) 1950.

34. Whalen, E. J.: Magnet Extraction of Foreign Bodies, Ann. Otol. Rhin. & Laryng. **59**:717 (Sept.) 1950.

35. Moersch, H. J., and McDonald, J. R.: Bronchial Adenoma, J. A. M. A. **142**:299 (Feb. 4) 1950.

36. Williams, R. B., Jr., and Daniel, R. A., Jr.: Leiomyoma of the Lung, J. Thoracic Surg. **19**:806 (May) 1950.

posteriorly over the midportion of the left side of the diaphragm, while the only bronchoscopic abnormality was narrowing of the left bronchus at the lower-lobe orifice.

Whiteside [37] reported a case of hamartoma of the lung in which the bronchoscopic findings were negative.

A case of solitary pulmonary xanthoma was reported by Ford and co-workers.[38]

Cases of fibrosarcoma of the bronchus were reported by Black,[39] Carswell and Kraeft,[40] and Curry and Fuchs.[41] In the latter case the tumor was expectorated, and there has been no recurrence in the four-year follow-up period. The importance of microscopic examination of coughed-up material was reemphasized.

BRONCHIAL CYTOLOGY

In an analysis of 307 consecutive cases of primary bronchogenic carcinoma, O'Keefe [42] pointed out, first, that the most recently developed diagnostic procedure, the cytologic study of bronchoscopically removed secretions, was a method that elicited a high percentage of positive results and added an appreciable margin of positive results in otherwise negative cases. Secondly, using the presence of carcinomatous mediastinal and/or bronchial nodes as criteria of resectability, he further showed that by obtaining diagnoses sufficiently early in the course of this disease, the possibility of operability was increased and a more favorable prognosis was indicated.

Woolner and McDonald [43] examined cytologically more than 400 patients with bronchogenic carcinoma. The false positive error was approximately 2%, and the false negative error was approximately 30%. Of 80 consecutive surgically removed bronchogenic carcinomas, 10% were peripherally placed and showed slight or no gross communication with the bronchial tree. Cytologic examination in the cases of these peripherally located tumors gave negative results. The authors concluded that cytologic studies were of value in the diagnosis of alveolar-cell tumors but of no value in bronchial adenoma or cylindroma.

Ross [44] described the results obtained in the diagnosis of bronchogenic carcinoma by cytologic study. A total of 178 specimens of sputum and bronchial secretions were examined by the smear and paraffin-section technique. Positive results were obtained in 17 of 20 proved or probable cases of lung cancer. In 90 cases apparently without pulmonary tumor one false positive report was made.

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37. Whiteside, J. H.: Hamartoma of the Lung, Canad. M. A. J. **63**:383 (Oct.) 1950.

38. Ford, W. B.; Thompson, C. W., and Blades, B.: Xanthoma of the Lung, Postgrad. Med. **8**:48 (July) 1950.

39. Black, H.: Fibrosarcoma of the Bronchus, J. Thoracic Surg. **19**:123 (Jan.) 1950.

40. Carswell, J., Jr., and Kraeft, N. H.: Fibrosarcoma of the Bronchus, J. Thoracic Surg. **19**:117 (Jan.) 1950.

41. Curry, J. J., and Fuchs, J. E.: Expectoration of a Fibrosarcoma, J. Thoracic Surg. **19**:135 (Jan.) 1950.

42. O'Keefe, J. J.: The Cytologic Diagnosis of Primary Bronchogenic Carcinoma, Laryngoscope **60**:931 (Sept.) 1950.

43. Woolner, L. B., and McDonald, J. R.: Cytologic Diagnosis of Bronchogenic Carcinoma, Dis. Chest **17**:1 (Jan.) 1950.

44. Ross, R. C.: The Value of Sputum Examination in Diagnosis of Cancer of the Lung, Canad. M. A. J. **63**:11 (July) 1950.

Tracheal aspiration of bronchial secretions was performed by Cahan and Farr [45] as a supplementary method of specimen collection for the cytologic diagnosis of carcinoma of the lung. With the patient under topical laryngeal and tracheal anesthesia, a small-caliber rubber catheter was blindly passed through the nose into the trachea, and secretions were secured by repeated flushing with saline solution and aspiration through the catheter. Preliminary efforts indicated that this method warrants further investigation.

ALVEOLAR-CELL TUMORS

In studying 12 cases of alveolar-cell tumors of the lung, Good and his associates [46] and Griffith and associates [47] found symptoms similar to those of other types of pulmonary disease, with cough a prominent feature and hemoptysis of frequent occurrence. The most valuable single aid in diagnosis was the cytologic examination of sputum or of bronchial secretions obtained through the bronchoscope. In the majority of instances this examination indicated that a neoplastic process was present in the lungs, and on some occasions a definite diagnosis of alveolar-cell tumor could be made.

BRONCHOESOPHAGEAL FISTULA

Pulmonary tuberculosis and primary bronchogenic carcinoma may simulate each other clinically and roentgenologically, and bronchoscopy often is the differentiating method of examination. In a case reported by Brackett and Lautz [48] two primary carcinomatous lesions were present, one in the mouth and the other in the esophagus, in addition to an associated pulmonary tuberculosis. The esophageal carcinoma had established a fistulous communication with the bronchus, but dysphagia was never present, the initial symptom being cough aggravated by ingestion of liquids and solids.

Coleman and Bunch [49] reported four patients with acquired nonmalignant esophagotracheobronchial fistula, all of whom had the characteristic symptoms. In treatment the authors recommended antibiotic therapy for fistulas associated with active tuberculosis or syphilis, local cauterization for small fistulas or those with exuberant granulations, and surgical treatment for the smooth epithelized communications or those with pulmonary complications.

Morton and co-workers [50] reported a case of congenital bronchoesophageal fistula with secondary bronchiectasis in an adult. Although cough had been present since

45. Cahan, W. G., and Farr, H. W.: Tracheal Aspiration: An Additional Method for the Early Diagnosis of Carcinoma of the Lung, Cancer **3**:475 (May) 1950.

46. Good, C. A.; McDonald, J. R.; Clagett, O. T., and Griffith, E. R.: Alveolar Cell Tumors of the Lung, Am. J. Roentgenol. **64**:1 (July) 1950.

47. Griffith, E. R.; McDonald, J. R., and Clagett, O. T.: Alveolar Cell Tumors of the Lung, J. Thoracic Surg. **30**:949 (Dec.) 1950.

48. Brackett, J. G., and Lautz, H. A.: Double Malignant Tumor Associated with Pulmonary Tuberculosis and Esophagobronchial Fistula, Arch. Otolaryng. **52**:225 (Aug.) 1950.

49. Coleman, F. P., and Bunch, G. H., Jr.: Acquired Nonmalignant Esophagotracheobronchial Fistula, J. Thoracic Surg. **19**:542 (April) 1950.

50. Morton, D. R.; Osborne, J. F., and Klassen, K. P.: An Apparently Congenital Bronchoesophageal Fistula Persistent to Adult Life, J. Thoracic Surg. **19**:811 (May) 1950.

infancy, the typical symptoms of congenital bronchoesophageal fistula did not appear until the patient had reached adult years and had previously been labeled "psychoneurotic."

ESOPHAGEAL BLEEDING

The prompt diagnosis of the cause of bleeding in patients with severe hemorrhage from the upper gastrointestinal tract is, at times, difficult, and Carter and Zamcheck [51] examined 14 patients esophagoscopically during the active bleeding phase without precipitation of further bleeding. Patients bleeding from esophageal varices must be distinguished from those bleeding from other lesions, because the prognosis and therapy depend upon the cause of the bleeding. The early diagnosis of varices in bleeding patients prevents unwarranted surgical intervention, while the exclusion of varices expedites prompt operation when indicated. Oxycel® gauze was kept available for direct application to bleeding varices, and in one patient esophagoscopy was performed solely for the therapeutic application of oxycel® gauze.

ESOPHAGEAL PERFORATION

Thomson [52] reported a case of spontaneous perforation of the esophagus which occurred in a histologically normal organ. The perforation was attributed to nausea with retching and vomiting.

Two cases of esophageal perforation following chronic nontuberculous empyema were presented by Oppenheim.[53] Conservative management and antibiotic drugs were used in the treatment.

CONGENITAL ESOPHAGEAL ANOMALIES

Congenital esophageal anomalies should be suspected when a newborn child has an excessive amount of mucus in the pharynx, feeding difficulties, and/or bronchopneumonia. Brown and Brown [54] reported 24 cases in a 10-yr. experience. The diagnosis was proved by obstruction to the passage of a catheter and by roentgen examination with an opaque medium. Surgical correction offers the only hope of survival and should be done within the first few days of life.

Belsey and Donnison [55] reviewed 10 cases of congenital esophageal atresia and pointed out that an infant that brings up an undue amount of mucus for several hours after birth should be suspected of atresia. Once the condition is thought of, the diagnosis can easily be established and prompt surgical treatment instituted.

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51. Carter, M. G., and Zamcheck, N.: Esophagoscopy in Upper Gastrointestinal Bleeding. New England J. Med. **242**:280 (Feb. 23) 1950.

52. Thomson, J. D.: Spontaneous Perforation of the Esophagus, Glasgow M. J. **31**:151 (April) 1950.

53. Oppenheim, H.: Unusual Etiology of Esophageal Perforation, Dis. Chest **17**:450 (April) 1950.

54. Brown, R. K., and Brown, E. C.: Congenital Esophageal Anomalies, Surg., Gynec. & Obst. **91**:545 (Nov.) 1950.

55. Belsey, R. H. R., and Donnison, C. P.: Congenital Atresia of the Esophagus, Brit. M. J. **2**:324 (Aug. 5) 1950.

ESOPHAGITIS

In studying experimental esophagitis, Ferguson and associates [56] have shown that the contact of acid gastric juice with esophageal mucosa has a prompt and destructive effect. The important factor was thought to be peptic activity, which requires a strongly acid medium, for hydrochloric acid alone had a comparatively mild effect upon the mucosa. The etiology of esophagitis was linked to vomiting, retching, or regurgitation.

Three cases of nonspecific granulomatous esophagitis were reported by Franklin and Taylor.[57] The roentgen findings in all three cases suggested carcinoma, but histologic examination of biopsy specimens showed granulation tissue. The authors compared this condition with regional ileitis.

ESOPHAGEAL STRICTURES

The problem of high esophageal stenosis was reviewed by Jackson.[58] High esophageal stenosis may be due to, in addition to carcinoma, cicatricial change, Plummer-Vinson syndrome (stenosis of the cervical esophagus, glossitis, hypochromic anemia), and fibrosis of unknown origin. In the presence of stenosis at this level esophagoscopy is difficult and dangerous unless a string or filiform bougie is first passed to act as a guide. In the management of this condition, the passage of perforated round metal dilators over the steel stem of a filiform bougie was recommended, so that subsequent passage of larger-sized metallic bougies could be done without delay.

Second-degree burns of the esophagus usually lead to stricture, and systematic treatment to prevent this complication is needed. Strumboff [59] advocated esophagoscopic bouginage in cases of a slight stricture and the continuous-string method with a bougie left in the strictured area constantly in the cases of severe stricture.

A preliminary study of patients with stenosis of the esophagus treated with an indwelling cannulated bougie was carried out by Tucker and his associates.[60] Although sufficient time has not elapsed for proper evaluation of the end results, this method of treatment was suggested in the following types of cases: acute lye burns, those cases in which the patients with cicatricial stenosis had not responded to other types of treatment, acute peptic ulcer, hiatal stenosis, and cardiospasm. In the technique, the bougie was suspended in the esophagus at the stenotic level for six to 12 hr., and the size of the bougie was increased as the stenosis widened. Care must be exercised to prevent laryngeal complications from pressure of the bougie upon the laryngeal cartilages, but this was avoided by suspending the bougie so that only the tip was in the hypopharynx.

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56. Ferguson, D. J.; Sanchez-Palomera, E.; Sako, Y.; Clatworthy, H. W., Jr.; Toon, R. W., and Wagensteen, O. H.: Studies on Experimental Esophagitis, Surgery **28**:1022 (Dec.) 1950.

57. Franklin, R. H., and Taylor, S.: Nonspecific Granulomatous (Regional) Esophagitis, J. Thoracic Surg. **19**:292 (Feb.) 1950.

58. Jackson, C. L.: High Esophageal Stenosis, Ann. Otol. Rhin. & Laryng. **59**:803 (Sept.) 1950.

59. Strumboff, A. V.: Chemical Burns of the Oral Cavity and Esophagus, Arch. Otolaryng. **52**:419 (Sept.) 1950.

60. Tucker, G.; Lautz, V.; Tracy, J. B., and Fearon, B. W.: The Diagnosis and Treatment of Benign Stenosis of the Esophagus, with Special Reference to Treatment by Dilatation with the Indwelling Cannulated Bougie, Ann. Otol. Rhin. & Laryng. **59**:823 (Sept.) 1950.

The role of abdominal infection in the production of benign esophageal stricture was discussed by Straus,[61] who reported two cases complicating duodenal ulcer. In one the stricture appeared after vagotomy and in the other after gastric resection. In both instances the condition responded satisfactorily to esophagoscopic bouginage and later peroral bouginage over a thread guide.

A complication not usually anticipated after lye strictures of the esophagus is the development of malignant degeneration in the scar. Of approximately 200 patients with esophageal lye burns seen by Bigger and Vinson,[62] carcinoma developed at the site of the stricture in seven. Since most of these patients were young children when the diagnosis was made, the question was raised whether the development of carcinoma in other members of this group might occur within the next few decades.

A case of benign esophageal stricture of 26-yr. duration in which carcinoma developed in the scar was reported by Redish.[63] Five months after surgical excision of the strictured area, metastasis to a previously normal trachea developed.

VAGOTOMIZED ESOPHAGUS

The esophageal aspects of vagotomy were studied by Friedberg [64] on a group of 31 patients. After vagotomy the prominent complaints were pyrosis, substernal discomfort, eructation, regurgitation, dysphagia, and excessive salivation. Despite the presence of symptoms, the esophagoscopic findings proved negative in some of the patients. In the patients with positive endoscopic findings, the most constant finding was excessive secretion in the esophagus. Temporary obstruction of the esophagus eventually resolved spontaneously with the use of warm liquids before taking solids, and mechanical dilatation was not necessary. Hiatal hernia, achalasia, and esophageal ulcer were observed as additional rare complications of the operation. The esophageal effects of vagotomy, as now performed, were considered to be of minor significance and offer no contraindication to the operation.

SYPHILIS OF THE ESOPHAGUS

A case of syphilis of the esophagus in which the lower end of the esophagus was found, on esophagoscopy, to be completely filled by a pink, finely irregular, nonulcerative mass was reported by Hudson and Head.[65] Although the biopsy specimen after histologic examination was reported as stratified squamous epithelium, the long history of dysphagia, the positive serologic examination, and the response to antisyphilitic therapy suggested the diagnosis of tertiary syphilis of the esophagus.

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61. Straus, G. D.: Stricture of the Esophagus Associated with Operation for Duodenal Ulcer, Arch. Otolaryng. **51**:165 (Feb.) 1950.

62. Bigger, I. A., and Vinson, P. P.: Carcinoma Secondary to Burns of the Esophagus from Ingestion of Lye, Surgery **28**:887 (Nov.) 1950.

63. Redish, M. H.: Benign Esophageal Stricture and Carcinoma of the Esophagus, U. S. Armed Forces M. J. **1**:447 (April) 1950.

64. Friedberg, S. A.: Observations on the Esophagus Following Vagotomy, Ann. Otol. Rhin. & Laryng. **59**:751 (Sept.) 1950.

65. Hudson, T. R., and Head, J. R.: Syphilis of the Esophagus, J. Thoracic Surg. **20**:216 (Aug.) 1950.

ESOPHAGEAL FOREIGN BODIES

Rashid [66] presented a case of a 5-mo.-old infant with a metallic button in the esophagus. There was no dysphagia present, but respiratory distress was so great that tracheotomy was required before removal of the foreign body.

Lodgment of foreign bodies in the respiratory or alimentary tracts occur from dental accidents, and five cases were reported by Edwards.[67] Difficulty in the diagnosis has arisen in the last decade because of the construction of oral prostheses with the base and teeth made of plastic material (methyl methacrylate), which has a low degree of radiopacity. When porcelain teeth and metallic bars and clasps are used, they become readily discernible on the roentgenogram.

Latella and Jones [68] reported a case of an open safety pin in an 8-mo.-old baby and discussed the problem it presented. They concluded that watchful waiting is indicated in freely movable open safety pins in the stomach not causing distress and that the length of the waiting period rests with the surgeon faced with the individual problem.

Laff [69] reported a case of fishbone which had penetrated the esophageal wall and become lodged in the adjacent mediastinal structures. By following the preformed channel made by the fishbone, he was able to remove it successfully perorally.

After removal of a sharp bony foreign body from the pyriform sinus, Gutteridge [70] was able to demonstrate by barium roentgenograms an esophageal perforation with an abscess cavity extending into the superior mediastinum. The infection was controlled with the sulfonamides and penicillin, without surgical drainage.

Bender and Clayton [71] presented a case in which attempted passage of a stomach magnet resulted in a fracture of the magnet. Since the fractured fragment (2.3 cm.) was found to possess as much magnetism as the original magnet (5 cm.), it was concluded that a 2-cm.-long magnet would be a much safer instrument to employ in removing gastric foreign bodies.

ESOPHAGEAL HIATAL HERNIA

Clerf and associates [72] reported a series of 110 cases of esophageal hiatal hernia in which the diagnosis and indications for surgical treatment were emphasized. The combination of dysphagia and pain in some location suggested the diagnosis, although it should be considered in patients with vague, indeterminate symptoms in the lower part of the chest or the upper part of the abdomen. There was no characteristic clinical picture, and cardiac, respiratory, or other gastrointestinal

66. Rashid, A.: Atypical Case of Foreign Body in the Esophagus, Laryngoscope **60**:945 (Sept.) 1950.

67. Edwards, R. W.: Accidents of Dental Origin, Arch. Otolaryng. **52**:88 (July) 1950.

68. Latella, P. D., and Jones, D. H.: An Unusual Course of an Open Safety Pin in a Baby 8 Months Old, Ann. Otol. Rhin. & Laryng. **59**:254 (March) 1950.

69. Laff, H. I.: Fishbone in Mediastinum Removed by Esophagoscopy, Ann. Otol. Rhin. & Laryng. **59**:690 (Sept.) 1950.

70. Gutteridge, E.: Roentgenograms of Mediastinal Abscess, Arch. Otolaryng. **51**:272 (Feb.) 1950.

71. Bender, M. S., and Clayton, S.: Fracture of Stomach Magnet, Arch. Otolaryng. **51**:616 (April) 1950.

72. Clerf, L. H.; Shallow, T. A.; Putney, F. J., and Fry, K. E.: Esophageal Hiatal Hernia, J. A. M. A. **143**:169 (May 13) 1950.

diseases were simulated. Errors in previous diagnoses were common among patients in this series. An important factor in the diagnosis was the use of both roentgenography and esophagoscopy. In this series the majority of diagnoses were made by both methods; however, in 13 cases the diagnosis was made by esophagoscopy when the roentgenograms were not conclusive or suggested other conditions. Although stenosis was determined equally well by either method, ulceration at the esophagogastric junction was detected oftener by direct visualization. Conservative treatment (medical and mechanical) was carried out in 69% and surgical treatment in 31% of the cases. Patients with the following indications required surgery: (1) progression of symptoms with an increase in size of the hernia; (2) severe or recurrent hemorrhage from chronic or recurrent ulceration, and (3) incarceration or threatened strangulation of the hernia.

A series of 29 patients in the older age group with esophageal hiatal hernia was reviewed by Pickhardt and his co-workers.[73] In elderly patients with or without incarceration or obstruction, but with symptoms of pain, bleeding, pressure, or ulceration, phrenic-nerve interruption was practiced.

Four cases of carcinoma associated with thoracic stomach of the short-esophagus type were reported by Smithers.[74]

Dorfman [75] reported a case of carcinoma associated with an esophageal hiatal hernia. The carcinoma was located at the esophagogastric junction and originated from the thoracic pouch of the stomach.

Another case of carcinoma in a herniated gastric cardia associated with short esophagus was described by Dawson and Richards.[76]

ACHALASIA

The pathogenesis, diagnosis, and various forms of therapy of cardiospasm were reviewed by Guttman and Simon.[77] Their best results over a 10-yr. period were obtained by using the Mosher and Einhorn dilators for treatment.

Hoover's [78] intensive study of the problem of cardiospasm was concerned with the etiology, symptomatology, diagnosis, and treatment of this condition. Microscopic study of the seven surgical specimens did not provide any specific information concerning the etiology, but all specimens showed fibrosis and inflammation. It was thought that this process alone could produce a stricture or at least resistance to passage of food, with resultant hypertrophy of the musculature. When the resistance was not adequately compensated, the pressure within the viscus produced dilatation. The author advocated the abandonment of the terminology of cardiospasm or achalasia and suggested that the condition be designated as stricture of the esophagus, the location be stated, and any further description regarding the etiology

73. Pickhardt, O. C.; Rafsky, H. A., and Ghiselin, F. H.: Treatment of Hiatus Hernia in Older Persons, J. A. M. A. **142**:310 (Feb. 4) 1950.

74. Smithers, D. W.: The Association of Cancer of the Gastric Cardia with Partial Thoracic Stomach, Short Esophagus and Peptic Ulceration, Brit. J. Radiol. **23**:261 (May) 1950.

75. Dorfman, M.: Carcinoma Associated with Diaphragmatic Herniation of the Stomach, Radiology **55**:254 (Aug.) 1950.

76. Dawson, J., and Richards, H. G. H.: Carcinoma in a Herniated Gastric Cardia Associated with Short Esophagus, Brit. J. Radiol. **23**:270 (May) 1950.

77. Guttman, M. R., and Simon, M. U.: The Treatment of Cardiospasm, Eye, Ear, Nose & Throat Month. **29**:245 (May) 1950.

78. Hoover, W. B.: Cardiospasm or Achalasia of the Cardia, Ann. Otol. Rhin. & Laryng. **59**:766 (Sept.) 1950.

be added. The clinical course of these patients varied greatly. Since a patient with achalasia may be relatively free from symptoms for a long period, it was difficult to evaluate accurately any type of treatment. The treatment advocated by the author was dilation of the stricture or its surgical removal. Failures from dilation were attributed to failure to apply the proper amount of pressure in the area of the stricture for a sufficient length of time. In the author's experience accidents from dilation occurred in approximately 0.9% of the patients treated by this method and resulted from too great and rapid stretching. Surgical failures resulted because the stricture was not removed or recurrence of a stricture was not prevented. The surgical procedure of cardiectomy was advocated for those patients requiring surgery, but the necessity of dilations after cardiectomy emphasized the fact that this procedure is not entirely satisfactory.

Allen [79] reported two cases of cardioesophageal relaxation producing vomiting in infants.

A case of cardiospasm with thoracic stomach and congenital shortening of the esophagus was reported by Gutteridge.[80] The occurrence of the cardiospasm at this abnormal level in the thorax suggested that the process was due to spasm or lack of relaxation of the circular muscle of the esophagogastric junction, the cardiac sphincter, rather than a pathologic lesion of the diaphragmatic pinchcock or liver tunnel.

In contradistinction to the usual achalasia of the terminal esophagus, Asherson [81] described a similar condition occurring at the esophageal entrance, which he termed achalasia of the cricopharyngeal sphincter. Failure of the cricopharyngeal sphincter to relax ahead of the oncoming bolus of food was thought to cause this incoordination. Varying degrees of dysphagia unaccompanied with mechanical obstruction or stenosis were produced in the 10 cases reported. There was partial or complete failure of relaxation of the cricopharyngeal sphincter, in contrast to paralytic conditions of the esophagus, in which food rapidly entered the upper esophagus but became lodged at the aortic-arch level. Endoscopic examinations revealed no underlying cause for the dysphagia, and the diagnosis was essentially a roentgenologic one employing the use of pharyngograms. This condition was encountered in association with the following lesions: (1) complete and partial pharyngeal paralysis; (2) thyrotoxicosis myopathy, involving the pharyngeal muscles; (3) bilateral recurrent nerve paralysis, and (4) bulbar paralysis in the elderly, and after partial pharyngectomy.

COMPLICATIONS OF ESOPHAGEAL DILATATION

Hubbard and Leven [82] considered the use of the swallowed thread for esophageal dilatation to be contraindicated in the presence of an anatomic loop (enteroenterostomy) at any level in the alimentary tract. Two cases of strangulation of the bowel from this cause were reported. Dilatation by means of a radiopaque filiform and follower was suggested as an alternative in such cases.

79. Allen, R. P.: Cardio-Esophageal Relaxation, Radiology **55**:214 (Aug.) 1950.

80. Gutteridge, E.: Cardiospasm with Thoracic Stomach and Congenital Shortening of the Esophagus, Arch. Otolaryng. **51**:102 (Jan.) 1950.

81. Asherson, N.: Achalasia of the Cricopharyngeal Sphincter, J. Laryng. & Otol. **64**:747 (Dec.) 1950.

82. Hubbard, T. B., and Leven, N. L.: Esophageal Dilatation: A Contraindication to the Swallowed Thread, and an Alternative Method, Surgery **27**:126 (Jan.) 1950.

O'Leary and Burnett [83] reported a case with lacerations of the jejunum caused by a swallowed string which had been drawn into the jejunum by a broken gastrostomy-tube flange.

ESOPHAGEAL TUBERCULOSIS

Frew [84] reported a case of tuberculosis of the esophagus in which gradual dysphagia had been present for three weeks. On endoscopic examination an obstruction was found 1 cm. below the cricoid cartilage, which was due to a swelling of the entire wall of the esophagus. No lumen was visible, but biopsy specimens were taken from the lesion, the histologic report of which was tuberculosis. The response to streptomycin therapy was prompt, and after five weeks all symptoms and signs had disappeared.

ESOPHAGEAL TUMORS

In reviewing the pathology, diagnosis, and surgical treatment of esophageal tumors, Ireland and Bryce [85] were of the opinion that the careful examination and assessment of esophageal lesions were the responsibility of the esophagologist, but cooperation with the roentgenologist, pathologist, and surgeon was essential for successful management. The duties of the endoscopist should be to (1) furnish an accurate biopsy specimen for histologic diagnosis; (2) determine the position and extent of the tumor; (3) serve as a source of information about operability; (4) render postoperative assistance by bronchoscopic aspiration in order to reduce pulmonary complications; (5) make routine postoperative esophageal examinations and perform dilatations at the site of anastomosis, if necessary, and (6) remove many benign lesions through the esophagoscope.

A case of neurofibroma of the esophagus was reported by Engelking and associates.[86] The tumor presented a nodular appearance through the esophagoscope, but since the mucosa was intact over the mass, biopsy was not taken, the diagnosis being made after surgical removal of the tumor.

Several cases of leiomyoma of the esophagus were reported, by Daniel and Williams,[87] Chi and Adams,[88] Goldman and Masters,[89] Cornell and co-workers,[90] and Dugan and Meagher.[91]

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83. O'Leary, C. M., and Burnett, H. A.: Lacerations of the Jejunum by a Taut String, Surgery **27**:893 (June) 1950.

84. Frew, I. J. C.: Tuberculosis of the Esophagus, J. Laryng. & Otol. **64**:774 (Dec.) 1950.

85. Ireland, P. E., and Bryce, D. P.: Esophageal Tumors, Arch. Otolaryng. **52**:1 (July) 1950.

86. Engelking, C. F.; Knight, M. D.; Brauns, W. H., and Hershberger, L. R.: Benign Tumors of the Esophagus, Arch. Otolaryng. **52**:150 (Aug.) 1950.

87. Daniel, R. A., Jr., and Williams, R. B., Jr.: Leiomyoma of the Esophagus, J. Thoracic Surg. **19**:800 (May) 1950.

88. Chi, P. S. H., and Adams, W. E.: Benign Tumors of the Esophagus, Arch. Surg. **60**:92 (Jan.) 1950.

89. Goldman, A., and Masters, H.: Leiomyoma of the Esophagus, Arch. Surg. **60**:559 (March) 1950.

90. Cornell, N. W.; Shehadi, W. H., and Sharnoff, J. G.: Leiomyomas of the Esophagus, Surgery **28**:881 (Nov.) 1950.

91. Dugan, D. J., and Meagher, R.: Esophageal Leiomyomas, Arch. Surg. **61**:1066 (Dec.) 1950.

A case of dysphagia due to an epithelial inclusion cyst of the esophagus was reported by Swartz and Arrag.[92] The roentgenogram was reported as normal, but the diagnosis was made esophagoscopically after a piece of tissue had been removed for histologic examination from the lesion in the upper esophagus with the subsequent escape of fluid and collapse of the mass.

A case of ciliated epithelial cyst of the middle third of the esophagus, producing partial esophageal obstruction from extralumenal pressure, was described by Gledhill and Morrow.[93]

A case of rhabdomyosarcoma of the esophagus, in which a striated muscle malignant neoplasm was found in a zone of the esophagus where normally only smooth muscle is situated, was reported by Thorek and Neiman.[94]

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92. Swartz, M., and Arrag, F.: Mucus Cyst of the Esophagus, Ann. Otol. Rhin. & Laryng. **59**:565 (June) 1950.

93. Gledhill, E. Y., and Morrow, A. G.: Ciliated Epithelial Cyst of the Esophagus, J. Thoracic Surg. **20**:923 (Dec.) 1950.

94. Thorek, P., and Neiman, B. H.: Rhabdomyosarcoma of the Esophagus, J. Thoracic Surg. **20**:77 (July) 1950.

Abstracts of Current Literature

Ear

CORRECTION OF FACIAL DEFORMITY RESULTING FROM FACIAL NERVE PARALYSIS. S. KAPLAN, Eye, Ear, Nose & Throat Month. **29**:424 (Aug.) 1950.

A simplified method of correction of the deformity caused by facial-nerve paralysis is described. This method consists of 0.015-gauge tantalum wire introduced through a spinal needle and so anchored that the orbicularis oris and orbicularis oculi are tensed by these slings. The temporal muscle is used as an upper anchor. The method is proposed because of its simplicity and because it can be used as a temporary support in conjunction with facial-nerve repair or as a permanent correction when the facial nerve fails to respond.

JENNES, Waterbury, Conn.

NEOMYCIN IN THE TREATMENT OF OTITIS MEDIA IN INFANTS. A. M. LAZAR and J. FISHMAN, Eye, Ear, Nose & Throat Month. **29**:484 (Sept.) 1950.

Lazar and Fishman describe a syndrome encountered in infants under 3 months of age. The children are poor feeders, restless, poor sleepers, cry a great deal, and do not gain any weight. There is a low-grade temperature elevation with an associated diarrhea. A thorough examination reveals aural signs of otitis media; often there is a red bulging eardrum membrane. Paracentesis provides immediate relief. The culture frequently reveals Pseudomonas aeruginosa. Often the nose and nasopharynx appear normal but on culture reveal the same organism as the ear. The usual otitis of infants can be treated very easily by the antibiotics now in use, but the middle-ear infection caused by Ps. aeruginosa presents a problem. The authors have experienced gratifying results with the use of a new antibiotic, neomycin, a preliminary report of which they present in this article.

JENNES, Waterbury, Conn.

THE CLINICAL DETECTION OF AUDITORY RECRUITMENT. P. DENES and R. F. NAUNTON, J. Laryng. & Otol. **64**:375 (July) 1950.

The clinical detection of recruitment is of value in distinguishing nerve deafness from conduction deafness and as a measure of cochlear function. In this paper an excellent description of recruitment is given, and the principles of recruitment tests are discussed.

The existing tests for recruitment are of two kinds. The first group is the loudness-balance type. The binaural loudness-balance test is reliable and simple but has the disadvantage of being applicable only when one ear is normal for the frequency tested. The monaural loudness-balance test is cumbersome and difficult to evaluate.

The second group of recruitment tests is based on the measure of the rate of change of loudness with change of intensity of the testing sound. These are the difference-limen tests. The tests of Bekesy and of Luscher and Zwislocki are based on this principle. Denes and Naunton have devised a new test of this type which they call the "D. L." test. In their test the intensity difference for loudness is determined by applying two sounds of the same frequency to the same ear and measuring the intensity difference between these sounds which is just perceptible as a change in loudness. The difference limen is measured at two intensity levels. If the difference limen remains the same or increases with the intensity, then recruitment is present; if it decreases, then there is no recruitment.

The recruitment tester used in the "D. L." test is described, as is the test procedure. A comparison is made between the "D. L." and loudness-balance tests, and the results agree very well. The advantages of this test are that it measures recruitment in one ear, it is simple to perform, and requires only five to 10 minutes to do.

LEJEUNE and LEWIS, New Orleans.

OTITIS MEDIA IN INFANCY: TREATMENT WITH STREPTOMYCIN. HUGH A. CARITHERS, J. Pediat. **36**:767 (June) 1950.

Carithers treated five infants with otitis media caused by Pseudomonas aeruginosa. All were under 20 weeks of age, and none were severely ill. Streptomycin was given to these infants

after penicillin and sulfonamides had apparently failed. On streptomycin administration they promptly recovered, even though the organisms in one case showed poor sensitivity to the drug in the laboratory.

CONGENITAL AURAL FISTULA. ROLAND B. SCOTT and CLINTON H. WOODING JR., J. Pediat. **36**:784 (June) 1950.

Congenital aural fistulas are thought to be the result of incomplete fusion of the six primary tubercles which in the embryo are destined to become the external ear. The condition has a hereditary nature. The commonest type is found at the anterior margin of the ascending limb of the helix. If a fistula becomes infected and suppuration occurs, the sinus tract must be excised. In uncomplicated cases the tracts may be obliterated by fulguration or sclerosing agents.

MACDONALD, Pittsburgh.

THE MANAGEMENT OF DEAFNESS. KENNETH M. DAY, Laryngoscope **60**:953 (Oct.) 1950.

Day reviews the general subject of deafness clearly and interestingly, with many sage observations. Particularly important are his remarks upon how to manage the psychological difficulties that arise from deafness. To quote, "From my own experience I have learned that the handicap of deafness is about 90% imaginary." Deaf persons would do well to read excerpts from the article.

HITSCHLER, Philadelphia.

EFFECT OF GUNFIRE UPON AUDITORY ACUITY FOR PURE TONES AND THE EFFICACY OF EARPLUGS AS PROTECTORS. FRED W. OGDEN, Laryngoscope **60**:993 (Oct.) 1950.

The effect of gunfire on hearing was studied in men who served from one to 14 months on firing ranges. The results of different types of firing and the effect of earplugs (various types) are separately evaluated.

Audiometric charts show the losses incurred for each man, the time of recovery, etc. There was a significant difference in hearing loss between those who did and those who did not wear ear protectors.

HITSCHLER, Philadelphia.

Nose

RHINOTOMY FOR EXPLORATION OF NASAL PASSAGES AND ACCESSORY NASAL SINUSES. JOHN E. BORDLEY, and WILLIAM P. LONGMIRE, Ann. Otol., Rhin. & Laryng. **58**:1055 (Dec.) 1949.

Occasionally, in the authors' experience, patients are found to have extensive lesions in the nasal passages and accessory sinuses which are difficult to treat by the usual intranasal surgical procedures because of inadequate exposure. The patients usually fall into one of the following categories: (1) those with intranasal tumors with extension to neighboring sinuses that require liberal exposure both for removal and for control of subsequent bleeding; (2) those who have had repeated and often inadequate intranasal surgery, with resultant scarring, stenosis, and loss of intranasal landmarks; (3) those having local osteomyelitis which requires increased intranasal exposure during surgery.

General anesthesia is given through an oral endotracheal tube. After appropriate intranasal mucosal incisions and elevations have been done, the frontal process of the maxilla is chiseled free up to the nasofrontal suture on both sides. An external incision, beginning on one side of the ala and extending up the nasomaxillary fold to the level of the nasofrontal suture, is then carried across the nasal bridge to the upper angle of the severed frontal process of the maxilla on the opposite side. The bony bridge is severed by chisel along the nasofrontal suture and, after separation of the septum at its base, the nose is reflected to the unincised side. Bleeding is controlled by ligature, coagulation, or packing.

The remaining intranasal portions of the septum and perpendicular plate of the ethmoid bone are removed. Exposure of the ethmoid, maxillary, and sphenoid sinuses is excellent. If exploration of the frontal sinuses is necessary, secondary incisions are made from the outer angles of the transverse glabellar incision along the eyebrow.

At the close of operation, the two portions of the septum are approximated with interrupted sutures of 0000 atraumatic chromic absorbable sutures. The external incisions are approximated subcutaneously with absorbable (surgical gut) sutures and externally with silk sutures. Application of dental compound stent and intranasal packing secures the external nose in position.

Subsequent septal perforation or necrosis of tissue on the reflected nose due to circulatory interference has not occurred in the cases in which this method of surgery has been used. The external cosmetic results are deemed satisfactory.

It is clear that the operation is indicated in only a few carefully selected cases in which the standard intranasal operations would not provide adequate exposure for the safe removal of an extensive lesion.

STEELE, Kansas City, Kan.

Miscellaneous

EFFECT OF STREPTOMYCIN ON TUBERCULOUS MENINGITIS. H. D. BRAINERD and H. R. EAGLE, Ann. Int. Med. **33**:397 (Aug.) 1950.

Twenty-seven patients with tuberculous meningitis were studied to determine the effect of streptomycin on the course of the disease. The intramuscular and intrathecal doses of streptomycin varied considerably. An attempt was made to treat all patients for five months by the intramuscular route. Intrathecal treatment was administered according to several plans.

The patients fell roughly into five groups, according to the course of the disease under treatment. 1. Nine patients died during the first four weeks of treatment without significant clinical or laboratory evidence of improvement. 2. Eight patients showed initial favorable clinical and laboratory response but later relapsed and died while still receiving treatment. These patients all survived more than eight weeks but died before the end of the five month therapy period. 3. Two patients showed initial favorable clinical and laboratory response but maintained slight persistent evidence of infection after completion of the five month therapeutic period. They died seven and 12 months, respectively, after the onset of meningitis. 4. One patient was free of all evidence of infection of the central nervous system after the completion of five months of treatment, but meningitis recurred 100 days later. 5. Six of the 27 patients are alive, from 18 to 31 months after the institution of treatment. Of the survivors, two manifest moderate residual neurological damage and a third has had three grand mal seizures but is otherwise entirely normal.

Brainerd and Eagle point out that most patients having a favorable response to treatment received more than 60 mg. of streptomycin daily per kilogram of body weight intramuscularly and over 2.0 mg. per kilogram daily by intrathecal injection. Toxic reactions to streptomycin are frequent at high dose levels but are a justifiable risk. In spite of current therapy, the prognosis of tuberculous meningitis must still be considered very grave.

ALPERS, Philadelphia [ARCH. NEUROL. & PSYCHIAT.].

FUNCTIONS OF THE PAROTID GLAND. LOUIS NASH, and LEWIS F. MORRISON, Ann. Otol., Rhin. & Laryng. **58**:976 (Dec.) 1949.

Reports appearing in the medical literature during the past 75 years have been reviewed in an attempt to get a clearer picture of the normal function and forms of the parotid gland. Many reports of functional abnormalities of the parotid gland also have appeared during this period.

The similar embryological origin of the parotid gland and other internal organs and the similarity of its function to that of the pancreas have stimulated considerable attention in the medical literature. In an attempt to find a carbohydrate-regulating hormone in the parotid, much animal experimentation and clinical observation have been done. The opinions of the investigators are at variance, but it seems agreed that there is a hidden, poorly understood factor in the parotid gland that has an effect on the circulating blood sugar. It is debatable whether this factor is a substance antagonistic to insulin or whether it has a pancreas-stimulating function.

The existence of asymptomatic enlargement of the parotid glands in association with other disease is unquestioned. The possibility that the enlargement is a functional hypertrophy to compensate for a deficiency of some other organ has been considered. No reported investigation has yet solved this interesting problem. In the laboratory investigation of affections of the

parotid gland, Nash and Morrison suggest, as an aid to diagnosis, the following procedures: (1) sialography as an aid to localization of obstructive calculi and for differentiation of various types of chronic infections; (2) cultures and microscopic examination of the parotid secretions; (3) studies on volume and rate of flow of the secretions of the quiescent gland; (4) determination of the salivary uric acid level as a means of measuring accurately the gland metabolism and function; (5) determination of the amount of salivary amylase in conjunction with serum amylase, and (6) biopsy as a means of differentiation between Mikulicz' disease and the many systemic, neoplastic, or chronic infective states that can cause a chronic symmetrical asymptomatic enlargement of the parotid gland.

STEELE, Kansas City, Kan.

NEBULIZED COCAINE AS AN ANESTHESIA FOR PERORAL ENDOSCOPY. H. FREDERICK KEIBER, and DAVID H. JONES, Ann. Otol., Rhin. & Laryng. **58**:1075 (Dec.) 1949.

Utilization of aerosolized cocaine solution as a topical anesthetic for peroral endoscopy is advised by Keiber and Jones. The necessary apparatus consists of an oxygen cylinder with humidifier, rubber tubing connecting the oxygen source, and a nebulizer. Wide rubber tubing, 8 cm. in length, is attached to the mouth end of the nebulizer, which contains 3 cc. of a 4% cocaine solution. Preoperative medication consists of oral administration of a barbiturate and hypodermic injection of morphine and scopolamine hydrobromide or atropine in proper dosage, according to age, body weight, and general condition of the patient. The patient holds the nebulizer in his mouth with lips closed around the rubber tip. Oxygen is released at a rate of 5 liters per minute. It is claimed that proper anesthesia usually is secured in 20 min. If supplemental anesthesia is necessary before bronchoscopy or laryngoscopy, up to 1 cc. of the 4% solution of cocaine is instilled by cannula on the vocal cords or into the trachea. Before esophagoscopy is performed, this supplemental application is made to the pyriform fossas.

The authors apparently have secured satisfactory topical anesthesia in 252 of 257 endoscopic procedures; in five cases general anesthesia was required. It is reported that patients accept the method well.

STEELE, Kansas City, Kan.

THE PHYSIOLOGY OF RESPIRATORY OBSTRUCTION. JOHN S. GRAY, Ann. Otol., Rhin. & Laryng. **59**:72 (March) 1950.

In otherwise normal persons who have an obstruction to respiration above the tracheal bifurcation, the first resulting physiological change is that of reduction in the pulmonary ventilation capacity. For example, an obstruction leaving an orifice half the diameter of the trachea has been found to reduce the ventilation capacity about 75% of normal, while an obstruction leaving an orifice one-fourth that of the trachea produces a reduction in ventilation capacity to 25% of normal. The second change in pulmonary physiology is hypoventilation, in which pulmonary ventilation fails to meet the body requirements for oxygen. As hypoventilation becomes severer, it induces, first of all, a rentention of carbon dioxide in the body, with a resultant increase in the free carbon dioxide in the blood. When measured as a rise in the arterial carbon dioxide tension, this is known as hypercapnia. Secondly, the oxygen saturation of the arterial blood is lowered, and arterial anoxemia develops. Thirdly, both hypercapnia and anoxemia combine to produce acidemia, or lowered blood pH. The blood changes of hypercapnia, anoxemia, and acidemia resulting from hypoventilation lead to asphyxia.

Pulmonary complications, such as infection, edema, partial blocking of bronchi or bronchioles, and atelectasis, intensify anoxemia and acidosis. The distress, apprehension, restlessness, disorientation, and uncooperativeness of a patient with respiratory obstruction probably are more apt to be due to hypercapnia than to anoxemia, which is relatively symptomless.

In early stages of respiratory obstruction, the asphyxial blood changes provide a powerful respiratory stimulus for the accessory muscles of respiration. However, if obstruction and hypoventilation continue, a point of crisis is reached at which respiration no longer is stimulated and the breathing effort weakens. A vicious cycle is established whereby self-accelerating asphyxia may terminate abruptly and fatally. The use of sedatives will only depress the respiratory centers and hasten this vicious cycle.

While oxygen therapy is important in the treatment of respiratory obstruction, it cannot correct hypercapnia, with its contribution to acidosis, or the pulmonary complications secondary to obstruction. Respiratory obstruction can be treated successfully only by removal of the obstruction and maintenance of the airway.

STEELE, Kansas City, Kan.

TREATMENT OF CARDIOSPASM. M. REESE GUTTMAN and MARSHALL U. SIMON, Eye, Ear, Nose & Throat Month. **29**:245 (May) 1950.

Cardiospasm is a neglected field of endeavor on the part of the average otolaryngologist today. The internist, surgeon, and psychoanalyst not infrequently fail in their efforts to effect a cure or subject their patients to ineffectual and at times disagreeable, or even heroic, procedures. Local dilatation provides a comparatively simple and nondangerous method of relief, provided the instrument is passed over a properly anchored thread guide. A simple technique is described which gives uniformly gratifying results and which uses rubber balloon dilators with hydrostatic or pneumatic pressure. The authors have used the Mosher and Einhorn dilators in 41 of a series of 43 cases of cardiospasm. One dilatation was necessary to effect lasting cure in 24, or 58 per cent. Another five, or 12 per cent, required two dilatations and the remaining 12, or 30 per cent, required three or more dilatations. The patients returned from time to time for dilatation administered in the office with the Hurst mercury bougie as minor dysphagia or discomfort developed.

JENNES, Waterbury, Conn.

MIKULICZ SYNDROME. SAMUEL I. KAUFMAN, Eye, Ear, Nose & Throat Month. **29**:257 (May) 1950.

The author presents a case of primary localization of Hodgkin's disease in the parotid and lacrimal glands which gave the appearance of Mikulicz syndrome. This preceded by almost three months the more typical manifestation which permitted the diagnosis of Hodgkin's disease to be made.

JENNES, Waterbury, Conn.

TECHNIQUE FOR STUDYING THE NASAL ABSORPTION OF ALLERGENS IN HUMAN BEINGS. R. A. CHAIT, and M. WALZER, J. Allergy **21**:153 (March) 1950.

In this study the authors utilize the established fact that a passively sensitized cutaneous site will react after the systemic absorption of a related antigen through various portals of entry. They employ the anterior part of the nasal septum as the site of application, using 1 sq. cm. cotton tabs and exactly 0.25 ml. of antigenic solution. With this method, they were able to prove that the rate of absorption in a given control, tested at long intervals, was fairly constant. The absorption rate from the septum appeared to be fairly representative of that from the anterior part of the respiratory nasal mucous membrane. Absorption of peanut antigen was demonstrated in 33 out of 34 subjects tested.

FRIEDBERG, Chicago.

ASTHMA WITH SINUS DISEASE. J. L. GUERRANT, A. MCCAUSLAND, and O. SWINEFORD, J. Allergy **21**:187 (May) 1950.

Treatment of asthmatic patients based on a combination of allergic management plus radical sinus surgery in carefully selected cases is advocated by the authors, from the University of Virginia. Patients are studied in an effort to obtain information regarding the identity of all offending allergens, the type and extent of sinus disease, the nature of all infecting organisms, the severity and duration of asthma, the status of the heart and lungs, and the causes of nervous tension.

A conservative regimen is begun as soon as possible, and many patients are thereby relieved. It is unusual to find such active sinus infection that surgery cannot be postponed for a few months, and most patients with asthma plus sinusitis should be treated conservatively for six to 12 months before resort is had to radical sinus surgery. Once the decision is made to operate on the sinuses, the surgery should be complete enough to remove all diseased membrane from all sinuses. Patients should not be subjected to extensive surgery until emotional factors have been considered and controlled if possible. A number of people will be benefited by psychiatric

management. Sinus surgery will not remove the necessity for continued allergic management in the atopic person.

Of 37 patients treated conservatively, 30 improved. Thirty-four were treated by radical surgery; 25 improved, and in 14 surgery seemed directly responsible for the improvement. Other factors may have been responsible in 11 of the patients. All 16 of the patients who failed to improve either cooperated poorly or had severe emotional disturbances. Surgical results were disappointing in patients less than 30 yr. old. Nonatopic patients had better results than atopic patients.

FRIEDBERG, Chicago

VENTILATORY TESTS IN BRONCHIAL ASTHMA. E. A. GAENSLER, J. ALLERGY **21**:232 (May) 1950.

A distinction is drawn between vital capacity (a measure of air moved irrespective of time) and maximum breathing capacity (a measure of air moved during a unit of time). In the past, the vital capacity has been almost universally used for the quantitative estimation of ventilation impairment due to bronchial asthma. Quantitative studies on patients brought out the fact that the vital capacity bears little or inconstant relationship to the ventilation insufficiency of bronchial asthma while the maximum breathing capacity appears to reflect this insufficiency with a high degree of correlation. The principal dynamic physiological manifestation of bronchial asthma consists of a reduction of the rate of flow of the expiratory air stream. The vital capacity measures air volume irrespective of expiratory time and is therefore an inadequate measure of the pulmonary insufficiency of bronchial asthma. The dynamic expression of the "time-volume" relationship is exhibited by the air velocity index. The mean index for 34 patients with bronchial asthma was 0.36, whereas the mean index for 150 patients with pulmonary insufficiency due to deficient parenchymal lung tissue was 1.23.

FRIEDBERG, Chicago.

FRACTURES OF THE MIDDLE THIRD OF THE FACE. R. R. STUTTS, J. M. A. Alabama **19**:267 (March) 1950.

The author describes an injury resulting from a severe motor accident. When seen three hours later, the patient had already received first aid, but further treatment was required to prevent the characteristic "dishface" deformity. The roentgenogram revealed not only comminuted fractures of the floor of both orbits but also of both nasal bones, the cribriform plate of the ethmoid, the base of both frontal sinuses, and the base of both malar bones. There were also fractures of other bones of the face. A high degree of edema of all the tissues was present, and reconstruction was done on the third day following the accident. The malar bones were elevated without surgical intervention; the maxilla was repaired by the use of multiple loop wire attached to the teeth, and at the same time rubber band traction was applied. After the fragments of the nasal bones had been molded ino place, they were supported by an intranasal splint provided with openings for the passage of air. The article is illustrated by photographs and roentgenograms of the field before and after operation.

SELTZER, Philadelphia.

CANCER OF THE FACE, MOUTH, AND NECK. L. T. BYARS, J. Missouri M. A. **47**:169 (March) 1950.

Byars presents a series of cases of carcinoma about the face by discussing their different characteristics and the various methods of choice in their treatment. Photographs showing the conditions before and after therapy illustrate the value of the methods chosen in these cases. As an example, a basal cell carcinoma of the nasal ala and adjacent lip was treated for 12 years, without cure, by repeated irradiation. A radical excision was then done and the defect finally repaired with a pedicle flap. The comment is included by the author that "it is much more important to achieve cure than to do plastic repair at the original operation, or too soon thereafter."

SELTZER, Philadelphia.

BRAIN TUMORS IN INFANCY AND CHILDHOOD. SIDNEY W. GROSS, New York J. Med. **50**:2267 (Oct. 1) 1950.

Working in the departments of Neurosurgery in Montefiore and Mt. Sinai Hospitals, New York, Gross made this report before the Medical Society of the State of New York at its annual

meeting in May, 1950. Olfactory or visual hallucinations may be the first sign of brain tumor in the very young. Vomiting and enlargement of the head in infants make the diagnosis relatively easy. In older children, headaches, failing vision, imbalance, and poor use of arms and hands should cause one to suspect cerebral lesion. One unusual instance is cited. A 2-mo.-old infant had the above symptoms for two days. Necropsy six weeks later disclosed "a large pineal tumor." Ventriculography "remains the most important diagnostic procedure in infancy and childhood." As to incidence, Gross states, "About 80 per cent of all tumors of the brain in individuals under fifteen years are gliomas." Craniopharyngiomas comprise an important group in early life. They are congenital and arise from cell rests in the craniopharyngeal duct. Operative mortality is still high in the very young, but "older children do surprisingly well." Careful preoperative localization and better surgical technique have reduced postoperative mortality greatly. Abandonment of the two-stage operation has helped, and, as a result, in most clinics at present surgical mortality does not exceed 10%.

VOORHEES, New York.

MORTALITY IN HEAD INJURIES. ELDRIDGE CAMPBELL, ROBERT D. WHITFIELD, and VERNON T. GRIZZARD, New York J. Med. **50**:2273 (Oct. 1) 1950.

The data here brought together stem from services in neurology in the Albany (N. Y.) Hospital and from Albany Medical College. The paper was read before the Section on Neurology and Psychiatry at the 144th Annual Meeting of the Medical Society, state of New York, May 12, 1950.

Between 1935 and 1949, there were 718 head injuries observed by the three authors. Of the patients, 60 died, a mortality rate of 8.4 per cent. Therapy must be directed to treatment of shock, maintenance of a free airway, wound care, and constant alertness for latent hemorrhage and infection. Maintenance of a free airway is aided by suction of secretions from the mouth and nose and by the "coma position," with the patient lying on his right or left side, never on his back. Turning to change position every two hours aids postural drainage. Feeding is by duodenal tube, and aspiration of food particles is scrupulously to be avoided. Dehydration is prevented by fluid intake of 1,500 to 2,500 cc. daily. Débridement is done; the wound is freed of foreign matter; a depressed fracture is, of course, trephined; the dura is sutured, and 5,000 to 10,000 units of penicillin is left in the area, plus parenteral administration of penicillin. The temperature, and pulse and respiration rate are taken every half hour, and "changes" in the patient's condition are constantly checked. Sedation is limited; no opiates are permitted; the bladder of an unconscious patient is catheterized, and special nursing care is imperative. Uncorrected hematomas were thought to be the commonest cause of fatalities. "The majority of such patients will recover spontaneously, but from 5 to 10 per cent will die" in spite of all efforts.

VOORHEES, New York.

DIRECT OPERATIVE REMOVAL OF BENIGN MIXED TUMORS OF ANLAGE ORIGIN IN THE PAROTID REGION. J. B. BROWN, F. MCDOWELL, and M. P. FRYER, Surg., Gynec. & Obst. **90**:257-268 (March) 1950.

The main concern anatomically in parotid gland surgery is the relationship of the gland and the tumor to the facial nerve. Since most benign tumors of the parotid are found in the superficial lobe, a direct surgical approach is presented and the incision is planned for maximal exposure. The incision is started at about the hairline at the crus of the helix, continues down in front of the ear, over the tragus, around under the lobule, and then turns, anteriorly into the neck along a natural fold. This allows elevation of a flap of sufficient size to expose the entire gland and adjacent area. Nerve fibers are stimulated by acute pressure as they appear in the field, with resulting movement of the group of facial muscles involved. Dissection is carried out with small pointed scissors, and the tumor is carefully removed from the surrounding gland. In instances of carcinoma, the cancer field may be considered to include the regional lymph nodes and neck dissection should be done simultaneously. Since employing their method on 75 patients with benign mixed tumors of the parotid, the authors have had no traceable evidence of recurrence of the tumor and no injuries to the facial nerve (temporary or permanent).

FRIEDBERG, Chicago.

Book Review

The Audiology Clinic: A Manual for Planning a Clinic for the Rehabilitation of the Acoustically Handicapped. By Moe Bergman, Ed. D. Acta Oto-Laryngologica, Supp. 89, 1950. Pp. 107. Obtainable from The Audiology Foundation, 1104 S. Wabash Ave., Chicago 5.

Rehabilitation of the acoustically handicapped is a comparatively new field, for until recent years progress had been slow in gaining knowledge of the psychological aspects of the physical handicap and in developing medical, surgical, and educational methods for rehabilitating the hard-of-hearing person.

Because of the correlation of ideas and experiences of many specialized workers in the field of audiology, it is now possible to solve a large percentage of the problems of the deafened child and adult and make them independent, self-sufficient members of our society. Research of physicists, in commercial (hearing aid) and acoustic fields, research of the medical profession, and developments by the psychologists and the educational group of rehabilitation and teaching methods have crystallized many of the thoughts and ideas. Practical experience has proved the worth of these.

Dr. Moe Bergman, the author of this book, has had wide practical experience in this audiological rehabilitation work in both the Army and the Veterans programs and is the Non-medical Director of the New York Veterans Audiology clinic. Though much of this monograph specifically describes the Audiology clinic of the New York Regional Office of the Veterans administration, with which he is most familiar, it also has a great deal of information based on experience and research, not only his own but also that of others in various allied fields who have concentrated their undivided efforts toward the rehabilitation of the deafened. All the material represents results of practically proved successes.

The author discusses the ideal clinic versus the "modest" unit; planning the instructional program; designing an audiology clinic; planning equipment for the audiology clinic, with suggested sources and approximate costs of items of equipment in an appendix, and personnel and personnel requirements. There is a chapter on the audiology facilities for the rehabilitation of the acoustically handicapped in the United States. In his conclusion he deals with problems in the further development of audiology and the prevention of deafness.

This book should be extremely helpful to anyone interested in audiology and audiological clinics, for though the ideal clinic, such as is described, is seldom feasible, owing to lack of trained personnel and the expense of elaborate physical and electroacoustic equipment, the basic ideas and philosophies for the rehabilitation of the acoustically handicapped are the same whether the clinic or rehabilitation service is simple or elaborate.

Though any publication in as new a field as audiology necessarily contains facts and figures that may be out of date by the time the book is published, the facts and figures derived from research and experience are valuable to new workers in the field. This book is unique in containing detailed information that definitely would be helpful in planning a clinic for the rehabilitation of the acoustically handicapped.

News and Comment

MEDICAL MOTION PICTURES

A revised catalogue of motion pictures available through the Committee on Medical Motion Pictures of the American Medical Association is now ready. Copies will be sent to the secretary of each count and state medical society. This catalogue lists 62 16-mm. films, most of which are at the professional level. Fourteen of these films are suitable for showing to lay groups. Eight new films have been added. Copies are available upon request from the Committee on Medical Motion Pictures, American Medical Association, 535 No. Dearborn St., Chicago 10.

Directory of Otolaryngological Societies *

INTERNATIONAL

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

Meeting: Third Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology
Place: Habana, Cuba. Time: January, 1952.
President: Dr. José Gros. Information may be secured from Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

SECOND LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY

President: Prof. A. de Paula Santos.
Secretaries: Dr. José de Rezende Barbosa, Dr. Plinio de Mattos Barretto, Hospital das Clinicas, São Paulo, Brazil.
Place: São Paulo, Brazil. Time: July, 1951.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Carl H. McCaskey, 20 N. Meridian St., Indianopolis.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Place: Chicago. Time: June 11-13, 1952.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Derrick Vail, 700 N. Michigan Avenue, Chicago 11.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn
Place: Palmer House, Chicago. Time: Oct. 14-20, 1951.

AMERICAN BOARD OF OTOLARYNGOLOGY

Secretary: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Palmer House, Chicago. Time: Oct. 9-12, 1951.
Place: Royal York Hotel, Toronto, Canada. Time: May 13-16, 1952.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Herman J. Moersch, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 128 So. 10th St., Philadelphia 2.
Place: Royal York Hotel, Toronto, Canada. Time: May 23-24, 1952.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Royal York Hotel, Toronto, Canada. Time: May 20-22, 1952.

SECTIONS:

Eastern.—Chairman: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Place: Waldorf-Astoria, New York. Time: Jan. 11, 1952.
Southern.—Chairman: Dr. Lester A. Brown, 490 Peachtree St., Atlanta.
Place: Academy of Medicine, Atlanta. Time: Jan. 14, 1952.
Middle.—Chairman: Dr. Ralph J. McQuiston, 20 N. Meridian St., Indianapolis.
Place: Indianapolis Athletic Club, Indianapolis. Time: Jan. 28, 1952.
Western.—Chairman: Dr. Howard P. House, 1136 W. 6th St., Los Angeles.
Place: County Medical Building, Los Angeles. Time: Jan. 19-20, 1952.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Royal York Hotel, Toronto, Canada. Time: May 18-19, 1952.

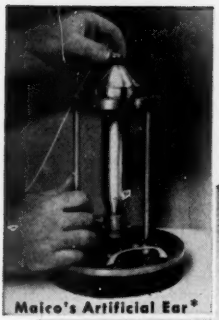
AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.
Secretary: Dr. Joseph G. Gilbert, 111 E. 61st St., New York 21.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. William H. Evans, 24 Wick Ave., Youngstown 3, Ohio.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.



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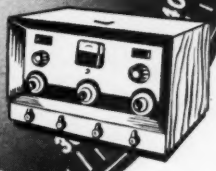

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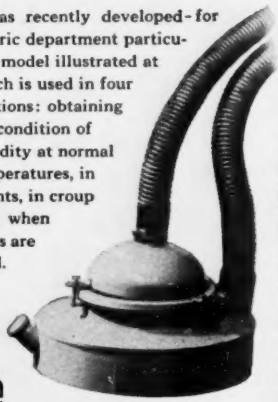

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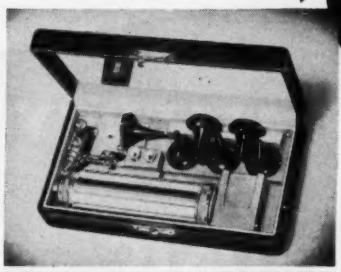

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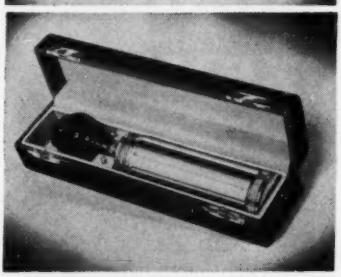

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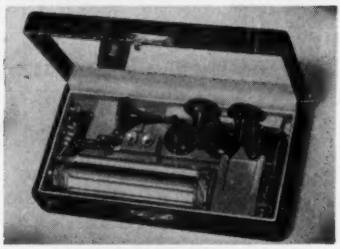

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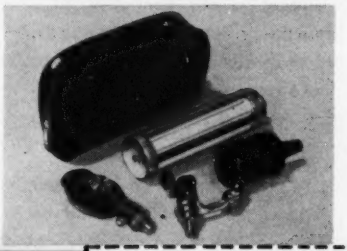

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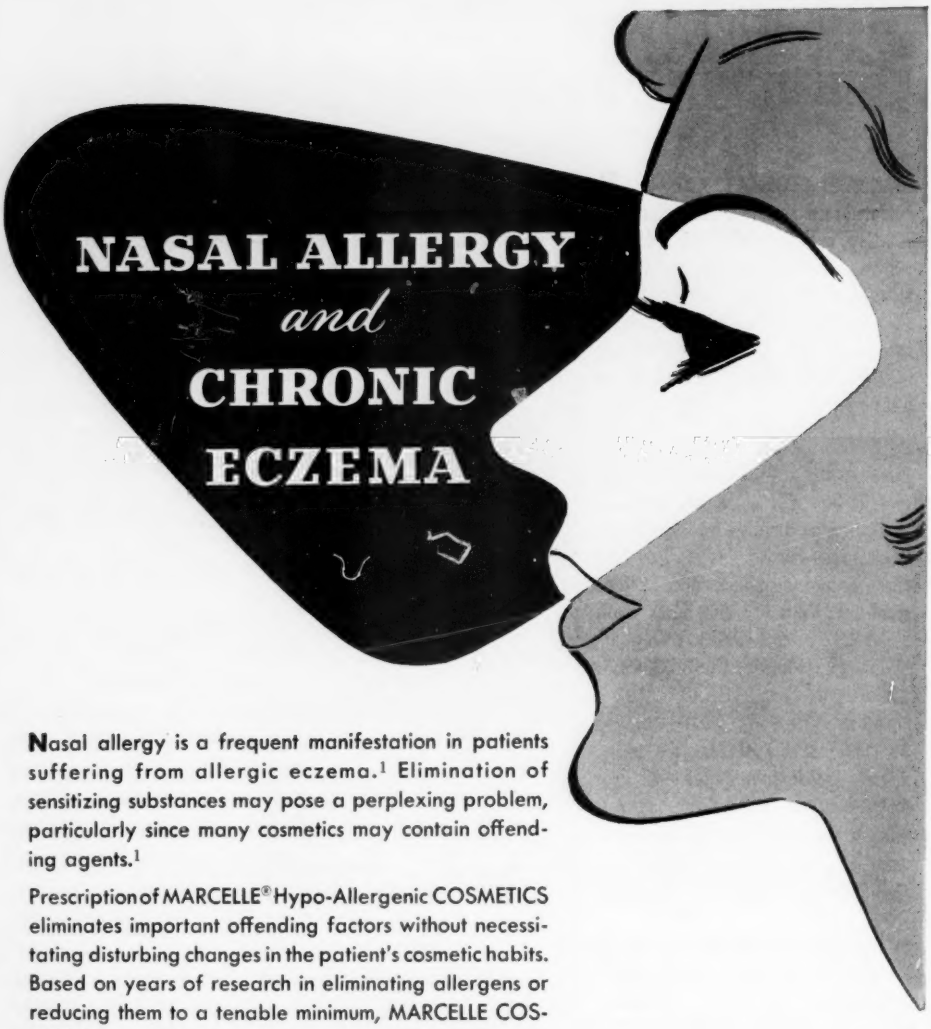

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1. Waldbott, G. L.: Management of Allergic Eczema. Am. Pract. 4:59-63 (Oct.), 1949.

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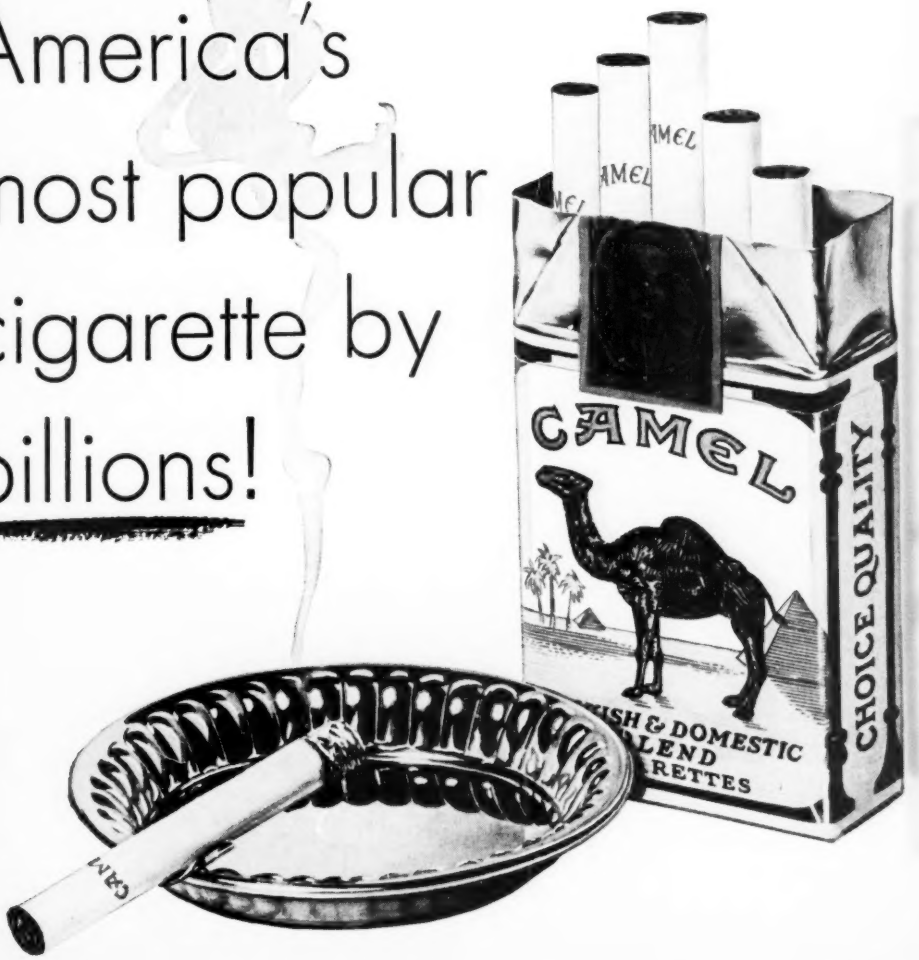
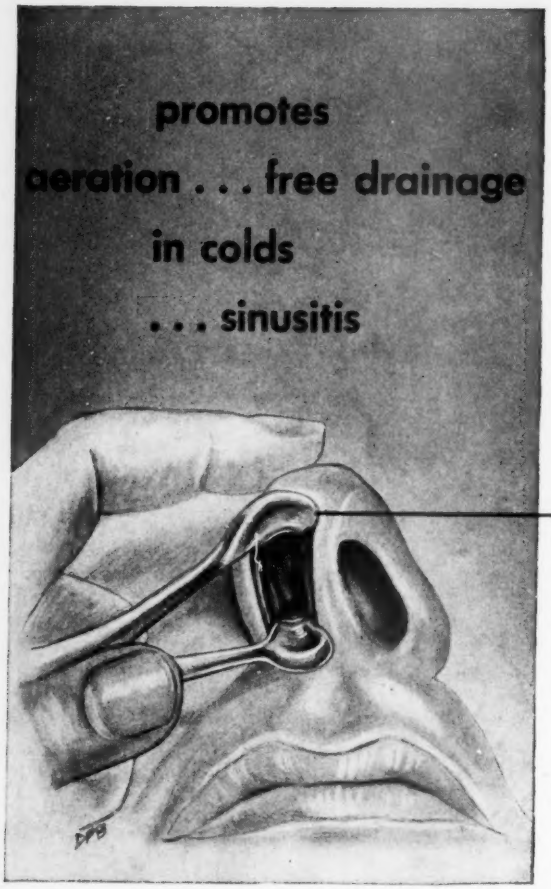
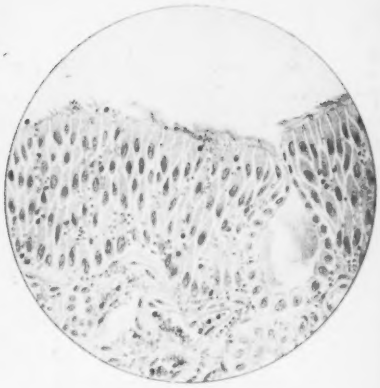
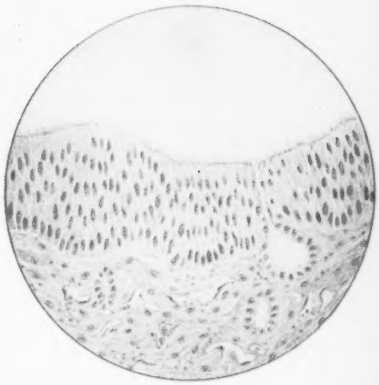






Nasal membrane showing increased leukocytes with denudation of cilia.

Normal appearing nasal epithelium.



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